Annual AVMA Meeting
Miami Beach, Florida
August 12-16, 1962

Journal

OF THE

AMERICAN VETERINARY MEDICAL ASSOCIATION

Myopathy in Hindlegs of Horses

A REPORT on effectiveness of surgical correction of this condition. Page 1089

Foot Rot in Sheep

DISCUSSION of a plan for control and eventual eradication of foot rot in sheep. Page 1093

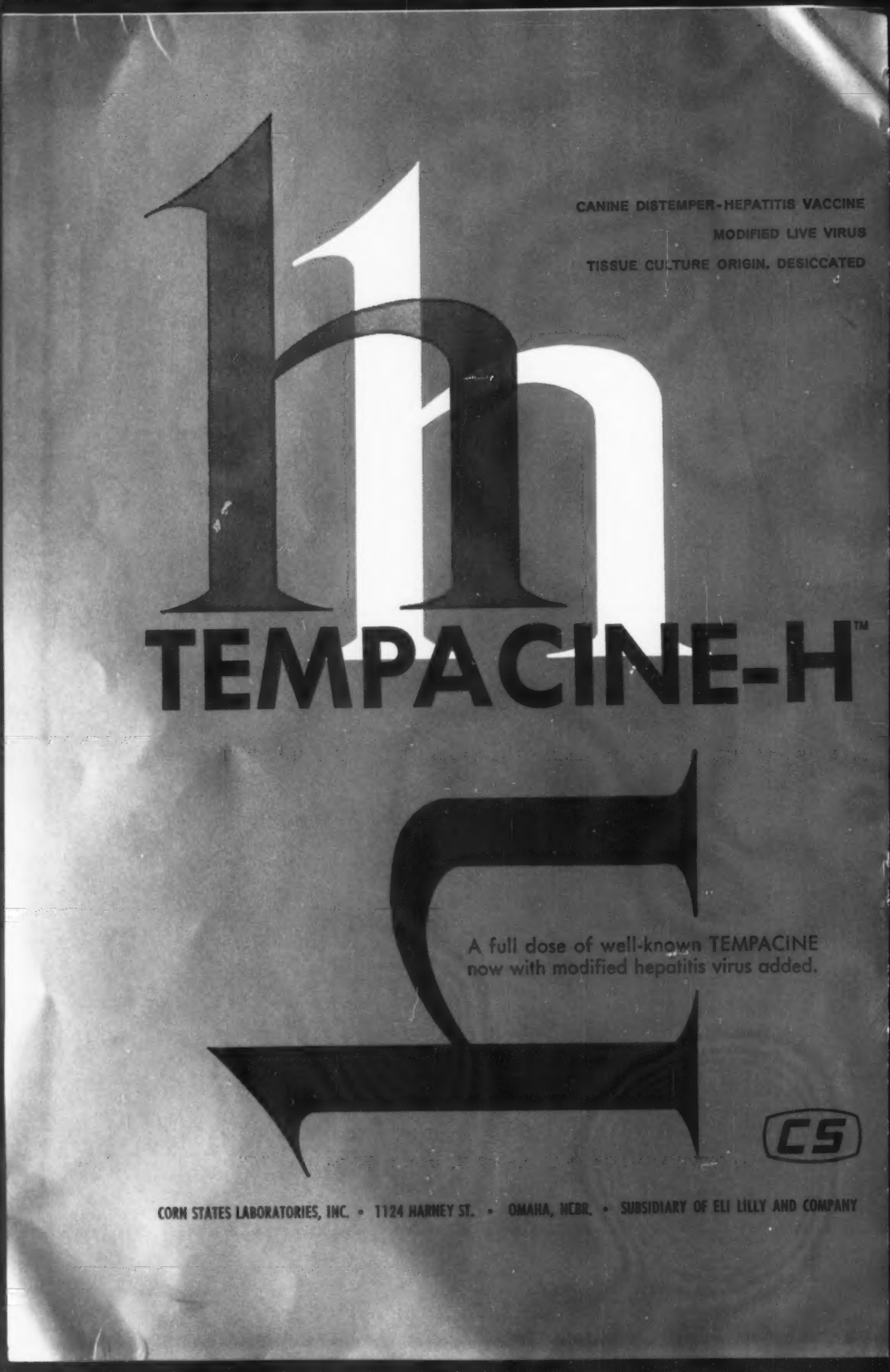
Injectable Anthelmintic for Canine Hookworms

DISOPHENOL was found to be an anti-hookworm agent effective when given subcutaneously, orally, or intramuscularly. Page 1101

Xanthomatosis in Chickens

OBSERVATIONS are reported on xanthomatosis in poultry during 1958-1960. Page 1106

Vol. 139 November 15, 1961 No. 10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Journal

OF THE
AMERICAN VETERINARY
MEDICAL ASSOCIATION

Vol. 139 No. 10 November 15, 1961

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Correspondence

Deigendesch

August 6, 1961

Dear Sir:

I was delighted to find upon opening my copy of the August 1 issue of the JOURNAL the wonderful article by Professor Cowen and your editorial in praise thereof! Such contributions along with the new format are continuing to make our JOURNAL a finer periodical.

I have corresponded with Professor Cowen since I possess a few copies of veterinary Americana, including a copy of the Deigendesh in his article. I am naturally proud that our State University (Rutgers, New Brunswick, N.J.) and Professor Cowen have made this veterinary contribution and I hope you can prevail upon them for another article soon.

Thanks again for printing such works. I do hope you are considering something on the role of our profession during the Civil War to coincide with our centennial in '63.

s/ROBERT R. SHOMER, V.M.D.
Teaneck, N.J.

Availability of Drugs Cited in Reports

September 5, 1961

Dear Sirs:

In the JOURNAL of Dec. 15, 1958, page 611, an excerpt from the *British Medical Journal* tells of successful treatment of bovine bracken poisoning with D- batyl alcohol.

From time to time when confronted with one of these more hopeless type ailments, I drive every drug supplier in the area beserk trying to obtain the proper drugs.

It would be most helpful, from the practitioner's standpoint, if these articles were footnoted so that one could know where drugs might be obtained, if obtainable, and at what price.

s/P. J. JONES, D.V.M.
St. Helens, Ore.

[ED. NOTE: Drugs mentioned in feature articles of the JOURNAL are identified in footnotes along with the manufacturer's name and address. However, availability of drugs reported in abstracts from other journals cannot always be determined, so we must rely on the reader's ingenuity to track down a source. Above mentioned agent is available from larger chemical suppliers at about $15 for 10 Gm.]

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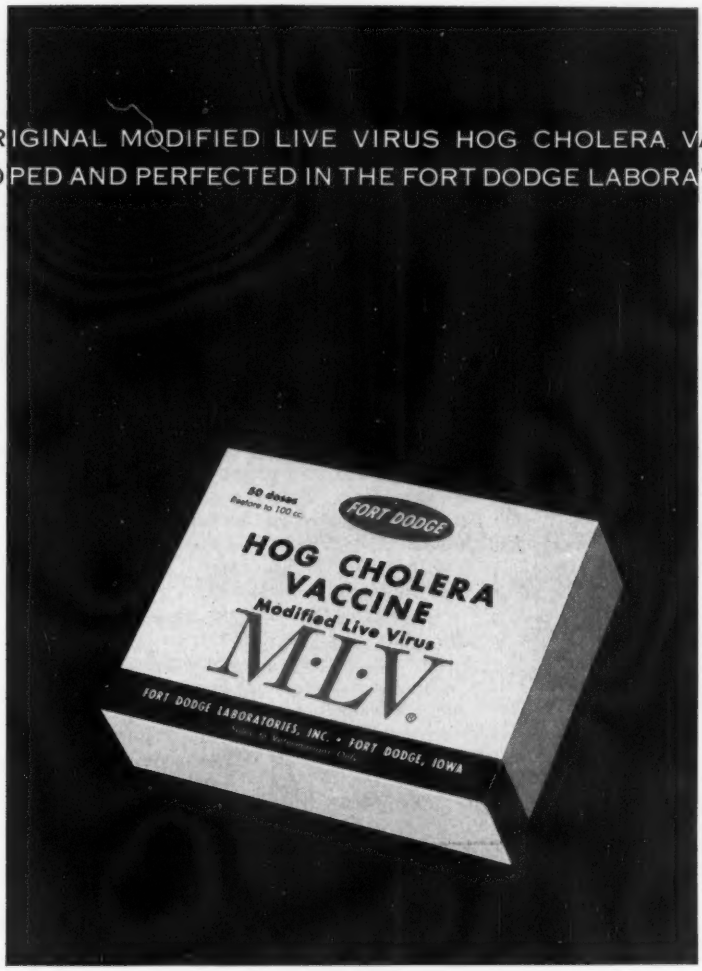

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news from Washington . . .

FROM THE AVMA WASHINGTON OFFICE
J. A. McCallam, VMD
Brig. Gen. USA (Ret.)

MILITARY

Veterinary Draft

The Defense Department has placed a call on the Selective Service System for 67 veterinarians (see JOURNAL, Oct. 1, 1961, p. 736), 495 physicians, and 154 dentists, beginning in November, 1961. Local draft boards have been requested by Selective Service Headquarters to prepare a special cover sheet, "Report of Availability and Summary Classification" (S.S.S. Form 129), for prompt submission; these are to be consolidated by each state director and forwarded to National Headquarters Selective Service no later than Oct. 18, 1961. Headquarters will of necessity resort to a draft call of veterinarians under the provisions of the Universal Military Training and Service Act as amended, unless the number now required are obtained voluntarily. Such a call, placed with all local draft boards, would have serious consequences on the veterinary profession.

Veterinarians who are draft vulnerable and in a position to volunteer for active duty, especially those in class 1-A and not yet 26 years of age, *are urged to apply without delay* for a commission in the Army Veterinary Corps. Direct your request for necessary forms and instructions to: Army Area Headquarters, army area in which you reside, Attention: AMEDS Personnel Counselor, Surgeons Office. The correct address of an Army area headquarters may be obtained from any military office or post office.

According to information received, veterinarians found fully qualified for a commission (or inducted) can expect to be on duty early in January, 1962, in the grade of first lieutenant.

LEGISLATION

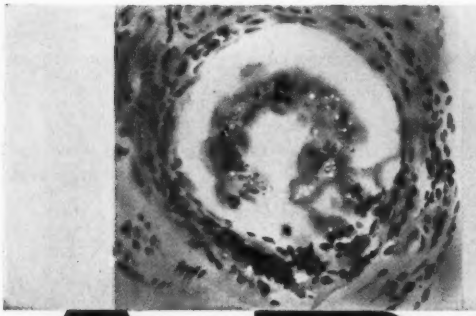
International Cultural Exchange

Public Law 87-256 (H.R. 8666)—Provides for strengthening of the international relations of the United States by promoting better understanding through educational and cultural exchanges with other countries; approved by the President, Sept. 21, 1961.

Peace Corps Act

Public Law 87-293 (H.R. 7500)—Provides for a Peace Corps to help peoples of interested countries and areas to

(Continued on page 1056)



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meet their needs for skilled manpower. An amount not to exceed $40,000,000 was authorized to be appropriated for fiscal 1962 to carry out the purposes of this act; approved by the President, Sept. 22, 1961.

Disarmament Agency

Public Law 87-297 (H.R. 9118)—Establishes a U. S. Arms Control and Disarmament Agency; approved by the President, Sept. 26, 1961.

Community Health Facilities

Public Law 87-395 (H.R. 4998)—Called the Community Health Services and Facilities Act of 1961; to assist in expanding and improving community facilities and services for the health care of aged and other persons; approved by the President, Oct. 5, 1961.

Income Tax Account Numbers

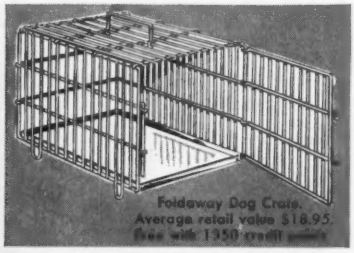
Public Law 87-397 (H.R. 8876)—Amends Internal Revenue Code of 1954 to permit the use of identifying tax account numbers to be included on income tax returns for proper identification. Effective date shall apply only to returns, statements, and other documents relating to periods commencing after Dec. 31, 1961; approved by the President, Oct. 5, 1961.

Defense Education Act Loans

Public Law 87-400 (H.R. 9053)—Amends Title II of the National Defense Education Act of 1958 with respect to the periods for which loans under that title are made. Loans will be for an "academic year or its equivalent, as determined under regulations of the Commissioner of Education"; approved by the President, Oct. 5, 1961.

HEW Appropriations

Included in HEW appropriations for fiscal 1962 is $23,000,000 to the Food and Drug Administration for plans, specifications, and construction of a special pharmacological-animal laboratory, with $1,750,000 to remain available until expended. Available to the Public Health Service will be $30,000,000, to be used for grants for construction of health research facilities, pursuant to Health Research Facilities Act of 1956 as amended by the Act of Aug. 27, 1958.



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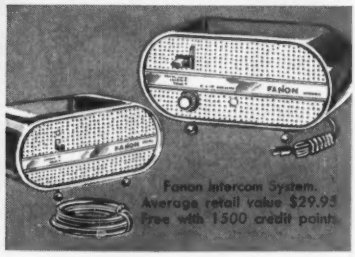

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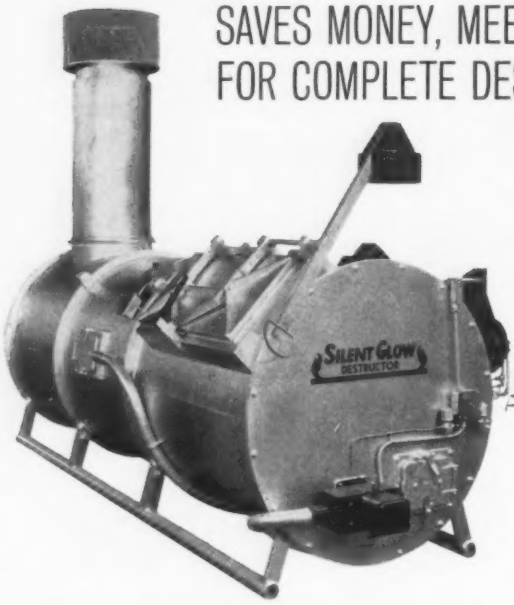

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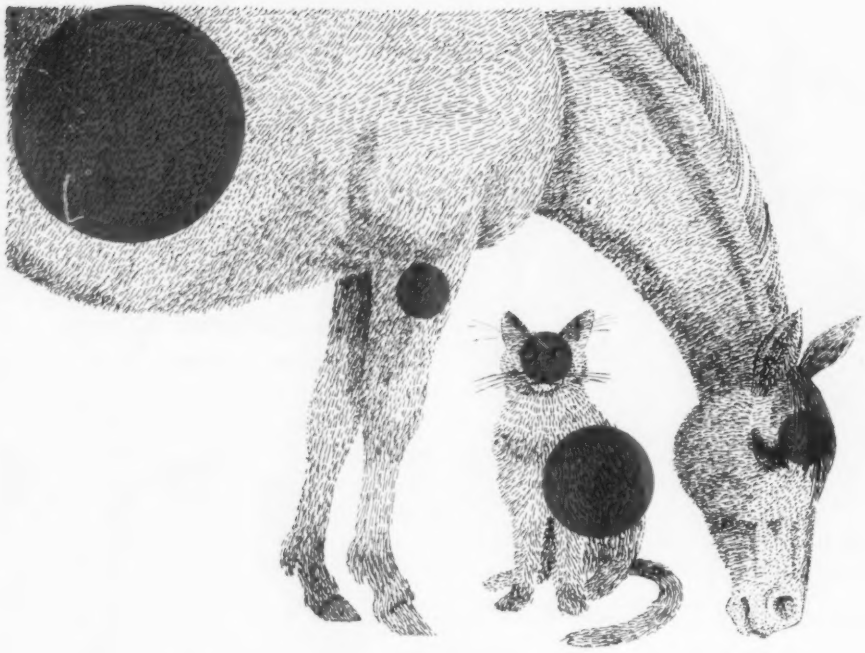

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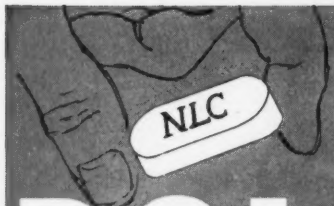

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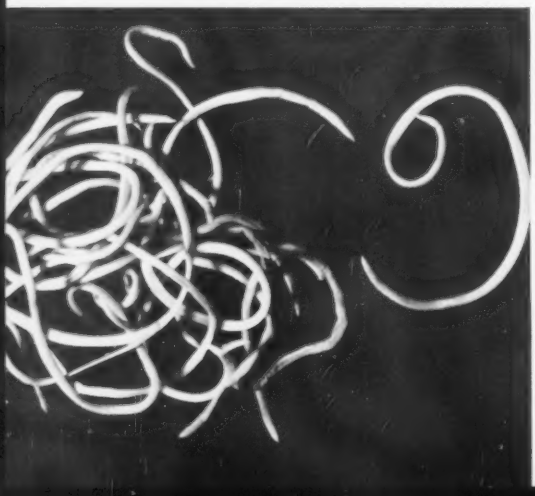
These are Roundworms or Ascarids from dogs
(Toxocara Canis)



Stable, Convenient and Effective

Extensive testing has established the safety, stability and effectiveness of MOM 'N PUP Dog Food—Medicated, even for puppies just starting to eat, and for bitches before and after breeding. It is approved for continuous feeding of dogs up to 18 months consecutively, and for use only upon the advice of a veterinarian.

The active worming ingredient in MOM 'N PUP is a stable active drug which has proved itself effective at levels low enough to be recommended for the treatment of both puppies and bitches. MOM 'N PUP eliminates roundworms as soon as they reach the adult stage in the intestines. Egg contamination is checked because few, if any, eggs are produced. MOM 'N PUP effects a natural movement and has no cathartic effects.

Inconvenient fasting, purging, and pill-forcing procedures common to ordinary anti-roundworm drugs are no longer required when a dog is fed MOM 'N PUP for roundworm control.

Non-toxic

Toxic signs associated with some medication, such as vomiting and diarrhea, are not observed with MOM 'N PUP. When dogs are fed at recommended, normal meal levels, they get a correctly established dosage—and they may be fed continuously or intermittently, depending on the extent of the roundworm problem.

Helps Repair Damage

MOM 'N PUP is equal in nutritional value to Kasco Dog Meal. It provides the complete

l of roundworms through n
pportunities for veterinaria

How a single $50.00 investment c
$480.00 per year for the veteri

Unique plan returns a 33⅓% profit

Ethical distribution of MOM 'N PUP Dog Food–Medicated provides additional earnings opportunities for the veterinarian. The small investment required for initial stock of MOM 'N PUP can be returned almost ten-fold in the first year.

A $50.00 investment by the veterinarian will purchase a basic inventory of 30 bags of MOM 'N PUP Dog Food–Medicated at the wholesale price tendered only to veterinarians. This is sufficient to treat or prescribe MOM 'N PUP for 30 dogs and puppies, on the average, in a two week period.

On an annual basis, using 30 dogs as the average number treated in a two week period – you can prescribe approximately

$1,200 worth of M
you a $480 profit
your practice enal
than a 30 dog aver
any case, MOM 'N
a 33⅓% return
profit that well ex
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The quick return
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Veterinarians
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Kasco repres
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nourishment needed to help repair worm-caused damage to a dog's health. MOM 'N PUP contains all the protein, fat, carbohydrates, vitamins and minerals that dogs are known to need for growth, maintenance and reproduction. Tests indicate that MOM 'N PUP Dog Food–Medicated has no effect on the reproduction process.

The palatability of Kasco MOM 'N PUP Dog Food–Medicated is excellent when mixed with water in the usual manner. Dogs will eat readily and take the medication without hesitation. MOM 'N PUP instantly releases an appetizing aroma –especially when mixed with warm water– and dogs eat with normal enjoyment.

**This three-way control also helps to protect families from the newly recognized danger to public health, visceral larval migrans – a serious and potentially fatal disease of children that follows ingestion of the infective eggs of the roundworms of dogs.*

MOM
'N
PUP
DOG FOO
MEDICAT
Available in
10 lb. and 25 lb. s

BEST FOODS DIV., Corn P

medicated
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t can earn
erinarian

of MOM 'N PUP. *This gives rofit annually!* And more if enables you to treat more average every two weeks. In M 'N PUP provides you with rn on your investment – a ll exceeds the return from bonds!
turn of your original invest- you to procure subsequent . And as you prescribe and N PUP, *your dollar-profit*

ans who do not dis- ay arrange with the presentative for local to handle MOM 'N PUP cription basis.

Corn Products Company, N.Y.

MAIL THIS CARD FOR COMPLETE INFORMATION

Veterinary Products Dept.
Best Foods Division, Corn Products Sales Co.
292 Madison Avenue, New York 17, N.Y.

Gentlemen:
Please send complete information on MOM 'N PUP Dog Food–Medicated

NAME_____

ADDRESS_____

CITY_____ ZONE_____ STATE_____

NAME AND ADDRESS OF YOUR VETERINARIAN_____

MAIL THIS CARD FOR COMPLETE INFORMATION

Postage Will Be Paid by Addressee

No Postage Stamp Necessary If Mailed in the United States

BUSINESS REPLY MAIL

FIRST CLASS PERMIT NO. 13813, SEC. 34.9, P. L. & R., NEW YORK, N.Y.

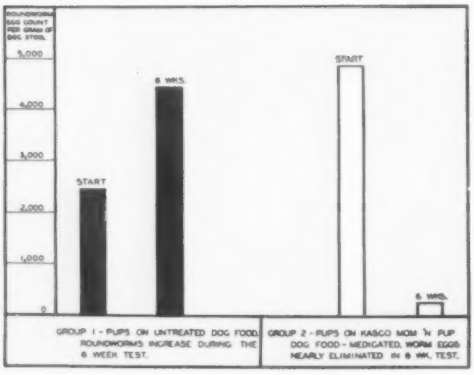
Veterinary Products Dept.
Best Foods Division, Corn Products Sales Co.
292 Madison Avenue, New York 17, N.Y.

Laboratory-Controlled Tests Prove Effectiveness of MOM 'N PUP Dog Food—Medicated

New Kasco MOM 'N PUP Dog Food–Medicated has been fully tested under strictly regulated laboratory conditions by a well-known pharmaceutical company, by the Kasco Research Laboratories, and clinically by practicing veterinarians. It is the only product of its kind available.

A controlled study of two groups of roundworm infested puppies, beginning at 7 weeks of age, demonstrated the remarkable effectiveness of MOM 'N PUP. One group of puppies was fed Kasco MOM 'N PUP Dog Food–Medicated. A second group was fed the same complete meal but without medication. After six weeks of continuous feeding, there was a marked difference in the clinical appearance of the two groups with respect to weight, hair coat and other health factors in favor of the medicated ration. Egg counts were performed on composite feces samples at the beginning and end of the test. Pups on *non-medicated* food showed marked increases in roundworm infestation. Those fed on Kasco MOM 'N PUP Dog Food–Medicated showed almost total elimination of roundworm eggs. These tests, shown on the chart, indicate that MOM 'N PUP promptly arrested roundworm infestation, and held roundworms under control during the test period.

PUPPY DEWORMING WITH KASCO MOM 'N PUP DOG FOOD–MEDICATED


ROUNDWORM EGG COUNT PER GRAM OF DOG STOOL
5,000
4,000
3,000
2,000
1,000
0
START
6 WKS.
START
6 WKS.
GROUP 1 - PUPS ON UNTREATED DOG FOOD. ROUNDWORMS INCREASE DURING THE 6 WEEK TEST.
GROUP 2 - PUPS ON KASCO MOM 'N PUP DOG FOOD - MEDICATED. WORM EGGS NEARLY ELIMINATED IN 6 WK. TEST.

Critical studies also were performed on mature dogs infected with Toxocara canis, Toxascaris Leonina, or both. The animals were fed daily with a ration of MOM 'N PUP Dog Food–Medicated. After a three-week period adult roundworms were almost completely eliminated, and egg production ended. A reduction in egg count in excess of 90% was observed in just one week. When infested dogs are fed Kasco MOM 'N PUP Dog Food–Medicated as directed, the ascarids are eliminated automatically as the roundworm develops into the adult stage in the intestine.

Q. *When fed continuously, what is the "accepted effective dosage" of diethylcarbamazine for ascarid control?*

A. From .7 to 1.0 milligram per pound of dog weight. The dosage received by dogs on Mom 'N Pup Dog Food-Medicated will vary between .7 and 2.0 milligrams per pound of dog weight depending on the effect of size and age on food intake.

Q. *What is the toxicity level of diethylcarbamazine?*

A. More than 30 times the level at which it is used in Mom 'N Pup Dog Food-Medicated for the control of ascarids.

Q. *Will Mom 'N Pup Dog Food-Medicated be useful in the control of filaria?*

A. No. Diethylcarbamazine has not been observed to affect adult heartworms at all. The level of drug in Mom 'N Pup Dog Food-Medicated is not high enough for perceptible effect on microfilaria.

Q. *Where and when has publication of experimental conclusions connected with the control of ascarids by diethylcarbamazine been made?*

A. Annals N. Y. Academy Science 50:141-160 (1948) • Journal A.V.M.A. November 1954 • Proc. Helminth Soc. Wash. 18:50-52 (1951) • Vet. Clinical Research Bul. June 13, 1955 • Journal of Lab & Clinical Medicine 33:216-234 (1948) • Lederle Vet Bulletin 13:(1)-13 (1954) • Annals Pharm. France 13:-539-556. 1955.

Council on Biologic and Therapeutic Agents Meets

Contamination of milk was again given major attention at the meeting of the AVMA Council on Biologic and Therapeutic Agents at the AVMA central office, Chicago, October 5 and 6.

At the meeting Dr. Fred J. Kingma (OSU '38), Washington, D.C., was requested by the Council to prepare a report on contamination of milk by biologic and therapeutic agents for publication in the JOURNAL. Problems of adulterated milk have been of concern to the Council for several years. In 1960 a special panel was appointed to study the matter and in the April 1, 1960, (page 295) issue of the JOURNAL a special report entitled, "The Problem of Adulterated Milk and How It Affects the Veterinarian," was published. At that time milk adulterated by penicillin was receiving the greatest amount of attention. Now that the penicillin problem is under control, efforts will be made to stop contamination of milk by other agents.

Council on Biologic and Therapeutic Agents members shown here are (left to right): Drs. Carl J. Norden, Jr., N. H. Booth, J. D. Ray, and James Saunders, Jr. Council members not shown in the pictures are Drs. Clyde Cairy, chairman; J. R. Harris; John E. Martin; and James H. Gillespie.

Procedures for handling live vaccines were also considered at the meeting. Dr. John M. Hejl (TEX '43), council consultant, was requested to prepare, for publication in the correspondence columns of the JOURNAL, an explanation of why the recent council report recommended the use of cold diluent for the reconstitution of desiccated vaccines.

Shown at work during the Council meeting are (left to right): Drs. Roger P. Link, vice-chairman; F. J. Kingma, technical consultant; J. M. Hejl, technical consultant; and Charles E. Bild, secretary.

The Advertising Committee of this Council considered and reported to the Council on the advertising policies of companies particularly in respect to apparent incompatibilities in advertising appearing in veterinary and lay publications.

Other committees of the Council on Biologic and Therapeutic Agents are the Committee on Biologics, the Committee on Standards and Toxicology, and the Committee on Therapeutic Efficiency.

The Council will meet again in Chicago on March 22 and 23, 1962.

Louisiana V.M.A. Elects New Officers

Officers elected by the Louisiana V.M.A. recently are: Drs. William J. Austin (AUB '53), Hammond, president; Robert R. Gibbs (OSU '49), New Orleans, vice-president; Robert B. Lank (KSU '42), Baton Rouge, executive secretary; and John K. Cooper (TEX '56), Baton Rouge, treasurer.

AVMA Council on Research Convenes

AVMA's Council on Research met in Chicago at the AVMA central office on Oct. 15 and 16, 1961. Here is a brief report on items considered at this meeting.

Study of Research Needs and Potentials

A proposal was made at the meeting that the Council support a special study of research needs and potentials. The Council recognizes the need and has supported the idea of planning for a study in depth of the utilization of the basic sciences in veterinary research at all veterinary institutions. Plans for this study are still in tentative stages with the view of obtaining outside support.

Euthanasia Study Panel

The Council has received a grant to help support a study on euthanasia of small animals. A panel of five veterinarians has been selected to analyze existing methods of euthanasia and make an appraisal of them. Acceptances of panel appointments have not yet been received.

Fund Raising for Fellowship Program

The Council expressed appreciation for the efforts and results of the Women's Auxiliary fund-raising campaign on behalf of the AVMA Fellowship Program. The fact that dues notice cards will be redesigned to give veterinarians an opportunity to contribute to the fund at the time they pay their dues was noted by the Council.

Fellowship Program Stipend

The Council took action to raise the AVMA Fellowship stipend to a maximum of $5,500. This increase is needed to keep the AVMA Fellowship stipend at a comparable level with other fellowships and coveted awards.

Fellows to Meet

A special meeting of all past and present AVMA fellows and past and present Council on Research members is being planned at a breakfast meeting to be held in Miami Beach, Fla., during AVMA's 1962 convention. The purpose of the meeting is to bring together all people who have been closely involved in the AVMA Fellowship Program to let them see the total impact of the program. The Fellowship Program has now been in existence for 20 years.

American Journal of Veterinary Research

The Council, through its Committee on the A.J.V.R., expressed approbation of format changes such as forward position of summaries, publication of interlingua summaries, publication of date manuscripts were received, and maintenance of high standards.

Expressing concern that advertising and subscription revenues do not quite offset production costs, the Council proposed that authors of research manuscripts should assist in defrayment of such costs.

The Council, whose members participated in the preparation of the *AVMA Style Guide* revision, was pleased to learn that the Guide has been well received. Since the initial mailing to colleges, experiment stations, government agencies, and publications, many requests have been received from AVMA members.

S. 1713 and H.R. 7293

The Council asked that AVMA oppose Senate Bill 1713 and House Bill 7293 which would create better pay scales in governmental positions for physical scientists than for biological scientists. The Council feels that the biological scientists in government

Members of the AVMA Council on Research in session here are (left to right around the table): Drs. J. T. McGrath, W. A. Hagen, R. D. Turk, C. R. Smith, T. C. Jones, R. Mitchell, Rue Jensen, C. A. Brandly, and G. Edds. Dr. G. C. Poppensiek, also a member of the Council, is not shown.

service should also get a better pay scale. The federal government's salaries of competent veterinary scientists are not at present competitive with those offered by universities and industry. As a result, the government cannot obtain all of the veterinary scientists it needs for its services. The Council does not object to the salary increases for physical scientists as such, but to the fact that the bills fail to meet the needs of some of the biological sciences. The Council reviewed the Miller Bill, H.R. 9454, and felt that this legislation should be supported by the AVMA since it appears to overcome the Council's objections to the two bills previously cited.

Future Scientists of America Program

Drs. Rue Jensen (COL '43) and George Edds (TEX '36) have been appointed judges for the next National Science Fair, International. The Council expressed appreciation to the AVMA Department of Public Information for its work on this student science fair in the past. It said it is important to inform people of the importance of humane care of animals used in science fairs, and pointed out that veterinarians are in an excellent position to work with scientifically-minded youngsters and to teach them respect for and humane treatment of animals.

AVMA Practitioner Research Award

The AVMA Practitioner Research Award is given to veterinarians who have completed worthwhile research while they were in active practice. The actual publication of that research work can be accomplished after a veterinarian has left practice and entered into other areas of activity. The Council, being responsible for selecting the winners, would welcome a larger number of nominations from the membership. The award is presented at AVMA annual meetings.

Grants for Meeting Attendance

Veterinarians who are seeking grants to attend international scientific meetings can apply to the AVMA Council on Research. The Council can sponsor such an applicant for a grant from one of the national granting agencies. However, application must be made one year prior to the scientific meeting the veterinarian wishes to attend; the meeting must be scientific in nature, not professional; and the applicant must be well known in his field.

Council Officers

Officers of the AVMA Council on Research are: Drs. T. C. Jones (WSU '35), chairman; George Edds (TEX '36), vice-chairman; and C. Roger Smith (OSU '40), secretary. The next Council meeting will be held March 17 and 18, 1962.

Volunteers Needed for Military Service

The U.S. Department of Defense has called for 67 veterinarians to report for duty in November, 1961.

This call is for 67 veterinarians in addition to those that will be called from graduating classes, and those in national guard and army reserve units called to active duty. If a sufficient number of veterinarians have not volunteered for military service by November 1, a general "doctor's draft" call is expected.

Unless the 67 required veterinarians have volunteered, veterinarians most likely to be affected by the general call up will be graduates of draft age who belong to no reserve or national guard unit and have not fulfilled their military obligation. Requests for deferment on the basis of community or personal hardship may be honored. If neither of these circumstances exist, these veterinarians are draft vulnerable.

[As this issue of the JOURNAL went to press, we learned that the Department of Defense had requested the Selective Service System to call 67 veterinarians for induction, beginning Nov. 1, 1961.—ED.]

Reservists Can Request Delay

Graduate veterinarians who are members of ready army reserve or national guard units can request to be screened out of the ready reserve on the basis of community or personal hardship, if either situation exists. They would be placed in the standby reserve. Veterinary medicine is on the Department of Labor list of critical occupations, so some veterinarians may be eligible for transfer out of ready reserve on this basis.

Veterinary students and faculty members who are members of the army reserve or

national guard units ordered to active duty can request delay, according to a memorandum sent by AVMA Washington representative, Gen. J. A. McCallum, to deans of veterinary schools. Delay in reporting for active duty is authorized for one academic year and renewal may be granted for the period the student is pursuing a full-time course of study. This regulation applies to graduates and undergraduates and enlisted and officer personnel. Students should request delay on DA Form 591, "Application for Delay to Active Duty," in duplicate. The form must be countersigned by the school official.

Students assigned to an army ready reserve unit should submit their forms through their unit commander. Nonunit personnel should submit them through the commander issuing the active duty orders. Students assigned to national guard should submit the forms to the State Adjutant General. Students holding ROTC commissions should send their request to the Commanding General of the Army Headquarters in which their college is located. Students in ROTC should submit their forms through their PMS & T.

Students who are not reservists and who have not been in service can secure deferments through their selective service boards.

Armed Forces Veterinary Officer Post-Graduate Training Program Initiated

A program has been established by the U. S. Department of Defense and the Selective Service System which will defer veterinary school graduates from active duty until they have completed their postgraduate training so that requirements of the military services for specially trained veterinarians may be met. It will become effective Jan. 1, 1962.

Under this program graduate veterinarians can apply for commissions in the Veterinary Corps Reserve of the Army or Air Force while they are completing postgraduate work at their own expense in civilian institutions.

The AVMA has agreed, in cooperation with the Department of Defense and deans of veterinary colleges, to furnish information and questionnaires to senior veterinary students and recent graduates of veterinary colleges. AVMA will also collect the completed questionnaires and forward them to the Department of Defense.

The Department of Defense will select applicants who will be offered deferments and allocate them to the military departments of their choice.

The Departments of the Army and Air Force will notify veterinarians of their selection in the program, offer them the opportunity to apply for reserve commissions, and take appropriate action to delay the call to active duty.

NSF Postdoctoral Fellowship Applications Available

Individuals who hold a D.V.M. degree and who desire to obtain further training for a career in research can apply now for a National Science Foundation postdoctoral fellowship. Deadline for applications is Dec. 18, 1961.

Fellowships will be awarded on the basis of ability only to persons who are citizens or nationals of the United States as of March 1, 1962, and who have demonstrated ability and special aptitude for advanced training in the sciences. A plan of study or research at the postdoctoral academic level should be submitted with the application.

Fellowships are awarded for scientific study and work at appropriate nonprofit American or nonprofit foreign institutions. All arrangements for admission to the institutions of the fellow's choice must be made by him.

The annual stipend for postdoctoral fellows will be $5,000. Fellows who receive awards for tenures of longer or shorter duration will receive stipends and allowances in proportion to the tenure of their fellowships. Married fellows normally will be provided an annual dependency allowance of $500 plus $500 for each dependent child. Fellows must agree not to receive remunera-

tion from another fellowship, scholarship, or similar award or federal grant or contract during the tenure of the fellowship.

Application materials may be obtained from the Fellowship Office, National Academy of Sciences—National Research Council, 2101 Constitution Ave., N.W., Washington 25, D.C. Fellowships will be awarded on March 15, 1962.

AVMA Assistant Director of Public Information Appointed

Mr. Charles E. Ramsay, Jr.

Mr. Charles E. Ramsay, Jr., has joined the staff of the AVMA as assistant director of public information.

Mr. Ramsay will have as his major responsibility the publishing of the new AVMA Newsletter which will be sent to constituent association secretaries and officers.

A native of Chicago, he graduated from Millikin University in Decatur, Ill., and joined the Homewood-Flossmoor (Ill.) Star as a news editor. After two years in the Army, including 13 months in Germany, Mr. Ramsay was a reporter for the Chicago Tribune, Seymour (Ind.) Tribune, and the Decatur (Ill.) Review before coming to the AVMA.

Mr. Ramsay is married and has one son. He lives in Chicago.

NSF Fellowship Applications Invited

Applications for National Science Foundation graduate fellowships for advanced study in the sciences will be accepted through January 5, 1962.

To be eligible for graduate fellowships, applicants must be citizens or nationals of the United States and have demonstrated ability and special aptitude for advanced training in the sciences.

Fellows will be selected on the basis of ability as attested by letters of recommendation, academic records, and other appropriate evidence of potential ability for scientific study or work. Applicants for graduate fellowships are required to take the Graduate Record Examinations. Their qualifications will be evaluated by panels of scientists appointed by the National Academy of Sciences-National Research Council. Final selection of fellows will be made by the National Science Foundation.

Stipends for National Science Foundation graduate fellowships vary with the academic status of the fellows. Students entering graduate school for the first time or those who have had less than one year of graduate study are considered first year fellows and will receive annual stipends of $1,800. Fellows who need no more than one calendar year of training to complete the requirements for the doctoral degree will receive annual stipends at the rate of $2,200. Fellows between these two groups will receive stipends at the rate of $2,000 annually. For married fellows a dependency allowance of $500 per annum for the spouse and a further $500 for each dependent child will normally be available. Tuition, laboratory fees and limited travel allowances will also be available.

Application material for National Science Foundation graduate fellowships may be obtained from the Fellowship Office, National Academy of Sciences-National Research Council, 2101 Constitution Ave., N. W., Washington 25, D.C. Awards will be announced on March 15, 1962.

American Association of Veterinary Nutritionists

The American Association of Veterinary Nutritionists met in Detroit on August 21 during the AVMA convention there.

Officers elected at the meeting are: Drs. Charles G. Durbin (UP '49), College Park, Md., president; S. H. Morrison (GA '54), Clinton, Iowa, president-elect; and J. H. Bailey (ISU '46), Atlantic, Iowa, secretary-treasurer.

At the meeting Dr. C. K. Whitehair (KSU '40) reported on "Protein Problems in Pigs." A panel discussion entitled, "What Can We Do at the Grassroots Level to Improve Our Service in the Nutrition Field?" was also held. Panelists were: Drs. James Bailey (ISU

'46), Atlantic, Iowa; Harry Geyer (OSU '36), Columbus, Ohio; John Herrick (ISU '46), Ames, Iowa; C. K. Whitehair (KSU '40), East Lansing, Mich.; and R. O. Anderson (COL '35), Elkhorn, Wis.

National Association of Extension Veterinarians Meets

The National Association of Extension Veterinarians held their annual meeting on Aug. 20, 1961 in conjunction with the AVMA convention in Detroit.

Subjects discussed at the meeting were problems relating to specific pathogen free pigs in various areas; the new statewide plan of work for extension in use in some states; the team approach to extension presentations; contract veterinary practice; the Indiana airborne classroom with possibilities of use for professional short courses and other communications; cow pool trends; farm livestock leasing; development of farm livestock auctions; large scale veal feeding operations; specific antibody milk production; trends in dairy herd size, numbers, and production; and hog cholera control progress.

Officers of the association are: Drs. William Brown (COL '44), Ft. Collins, Colo., president; Crosby Howe (COL '29), Lincoln, Neb., vice-president; and Wayne Burch (KSU '37), Madison, Wis., secretary.

Veterinary Film Wins Award

A veterinary film illustrating a new surgical technique was named the best professional medical class film of the year by the Biological Photographic Association at its recent meeting in Chicago.

Entitled "Respiratory Problems in Brachycephalic Dogs," the film shows Dr. Harmon C. Leonard (COR '44), Cheshire, Conn., performing surgery to correct obstructed air passages involving the nostrils, soft palate and larynx. Dr. Leonard also narrates the film which was produced for Schering Corporation's Veterinary division by Campus Film Productions, Inc.

Eight hundred entries from the United States and 22 other countries were made in all categories of the international competition. Medical, dental, veterinary, research, and other scientific organizations were represented. The winning film was judged for its photographic quality, achievement of purpose, and total competence in production.

"Respiratory Problems in Brachycephalic Dogs" is a 16 mm. color film with a running time of 22 minutes. It is available to interested veterinary groups on a lending basis. Requests should be made to Audio-Visual Department, Schering Corporation, 1011 Morris Ave., Union, N. J.

Chicago V.M.A. Raises More Than $5,000 for Fellowship Program

The Chicago V.M.A., in a recent special drive to augment funds raised by the Women's Auxiliary, raised more than $5,000 for the AVMA Fellowship Program.

A breakdown of Chicago area contributors looks like this:

Veterinarians	83	$4,212
Commercial Contributors	5	825
Private Contributors	1	300
Total		$5,337

No national firms were asked to help this fund drive. Only local companies were contacted.

Deaths

Star indicates member of AVMA

Charles F. Barnes (GR '15), 73, Anoka, Minn., died Feb. 12, 1961.

Dr. Barnes entered the meat inspection service of the USDA in January, 1929. He retired in December, 1954.

L. D. Belknap (CVC '15), 67, Knoxville, Iowa, died May 8, 1961.

Dr. Belknap was a general practitioner.

T. B. Clower (GA '33), 52, Douglasville, Ga., died of a heart attack Aug. 23, 1961.

Dr. Clower entered general practice in Valdosta, Ga., immediately after graduation. In 1941 he became chief veterinarian of the State Department of Agriculture. In 1954 he returned to general practice in Douglasville.

★**Leon A. Paquin** (MCG '98), 85, Northhampton, Mass., died July 24, 1961.

Dr. Paquin had been a milk, meat, and animal inspector and bacteriologist for Webster, Mass., for 62 years before his retirement two years ago. He became an honor roll member of the AVMA in 1957.

Women's Auxiliary

Committees Report Plans

Throughout the 1961-1962 Auxiliary year, the special emphasis on the work of all committees will be on broadening membership participation and increasing usefulness to the AVMA and the veterinary profession. Plans for the year are given here by committee chairman.

AVMA Fellowship Program Committee

Mrs. William G. Magrane

The outcome of the 1960-1961 fund raising campaign has been applauded by the AVMA as the most tangible service performed by the Women's Auxiliary in recent years. The $51,000 collected has been used to support 11 fellows. This is just a beginning! The goal this year is to be $100,000, and the theme of the campaign, "Give one half an average day's gross income for the future." The goal may sound staggering, but it can be reached if the story of the AVMA Fellowship Program is properly told by all Auxiliary members.

Veterinarians, like everyone else, hesitate to part with hard earned money—unless it's for a good cause. But if the Auxiliary can continually remind the profession that money contributed eventually increases their earning power, the task will be an easy one.

The 1961-1962 campaign is to be conducted by the members of the Auxiliary on a personal contact basis. Each state, through its executive board or secretary, will decide on the most suitable time to conduct a one month campaign. The 11 district directors are now at work securing helpers for the drive, and supplies are ready at the AVMA office.

Continuing education in research and teaching is a most important project in these changing times, and Auxiliary members can help their husbands' profession most through the work they do for the AVMA Fellowship Program for 1961-1962.

s/Mrs. William G. Magrane, *chairman*

Long Range Planning Committee

Mrs. Charles H. Ozanian

The function of this newly-created committee is to make recommendations to the Auxiliary's Executive Board and the House of Delegates for long range planning to achieve the Auxiliary's aims and purpose.

Here are some of the interlocking fields already suggested for consideration:

Financial responsibility—proportion of income for various activities and use of savings reserves.

Membership participation—areas in which this should be cultivated more.

Affiliated auxiliaries, constituent and regional—effective and mutually helpful relationships.

Auxiliary functions at AVMA conventions —purpose, participation, and scheduling.

Auxiliary structure—coordination with AVMA; responsiveness to members, to affiliates, and to AVMA; interrelationship of executive board, house of delegates, and members; auxiliary office.

Auxiliary usefulness to the AVMA and the veterinary profession—present areas and effectiveness, possible future areas.

s/Mrs. Charles H. Ozanian, *chairman*

Membership Promotion Committee

Mrs. Daniel Paradee

The membership is the life-blood of any organization. It is a constant challenge to encourage more new memberships as well as keep members we now have. Each year around 700 members are lost for non-payment of dues. The present Auxiliary membership of just over 8,000 is slightly over 50% of that of the AVMA. It is difficult to understand why every woman who has a sponsor is not an active member of the Auxiliary. She should be proud to be a member of the Auxiliary which is sponsored by the Association of her husband's profession. The dues are nominal—two dollars—and none of this money is used for entertainment.

The Auxiliary is prepared better than ever before to help constituents with membership campaigns this year. A leaflet telling "Why We Need You" is available for distribution and will be used on membership promotion drives. A brochure of general information is available from the Chicago office.

The wife, sister, or mother of every veterinarian on AVMA membership rolls is needed to promote veterinary medicine.

s/Mrs. Daniel Paradee, *chairman*

Nominating Committee

An honor roll point will now be granted if an affiliated auxiliary sends the names of women qualified for national AVMA Auxiliary work, either on committees or as officers, to the Nominating Committee.

Mrs. Wayne H. Riser

Both Mrs. E. F. Walker, of Oklahoma, and Mrs. V. J. Humphreys, of Wyoming, of the committee, have been conscientious workers in their own states and both have had experience as AVMA Auxiliary delegates. The committee feels that as many qualified women as possible should have a chance to participate and serve.

Therefore, affiliated auxiliary presidents should be urged to send the names and information about women recommended by their auxiliaries to this committee. This year a simple form to use as a guide in sending this information to the committee will be enclosed with the request for it.

s/Mrs. Wayne H. Riser, *chairman*

Public Relations Media Committee

Mrs. W. K. Wearly

The Public Relations Media Committee has many plans and projects in the works for the coming year. A blueprint of promotion ideas emphasizing the centennial celebration will be drafted soon.

The committee hopes to stimulate veterinarians and their wives to participate actively in science and health fairs in local schools. It will seek to promote an increased interest in public relations within student auxiliaries. It shall endeavor to continue the work being done in the area of recruitment.

All of these things add up to the real objective of this committee: to make every veterinarian and his wife aware of the many public relations opportunities which exist in every community. The success or failure of any public relations program depends directly upon the amount of participation, on the local level, by individual members of the profession.

s/Mrs. W. K. Wearly, *chairman*

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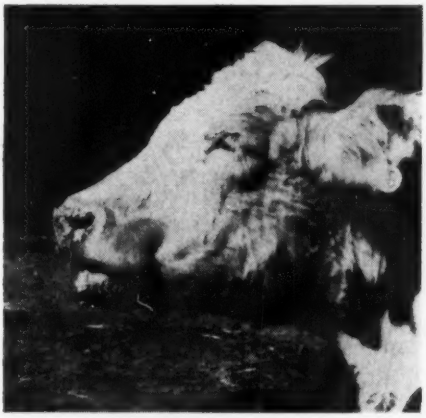

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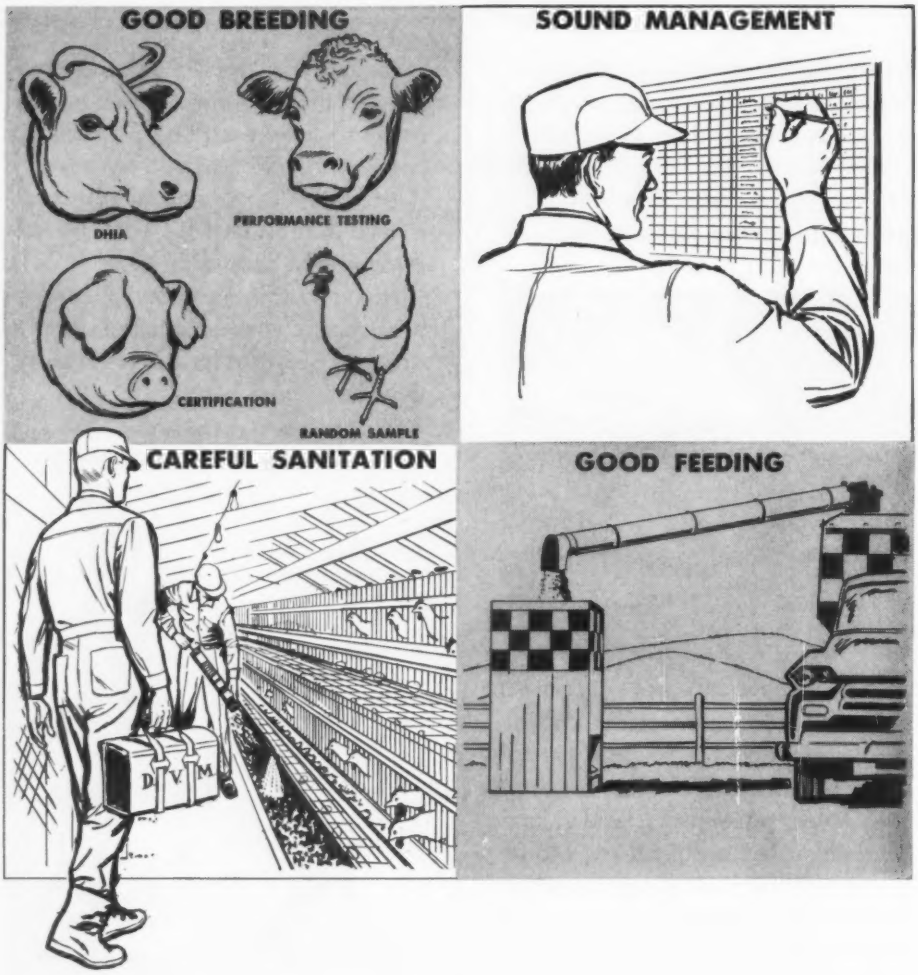

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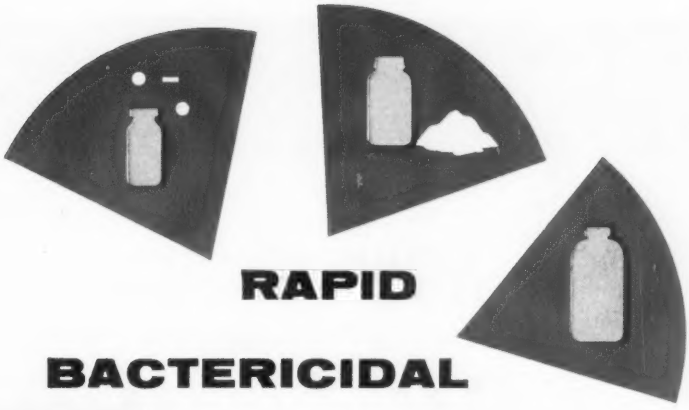

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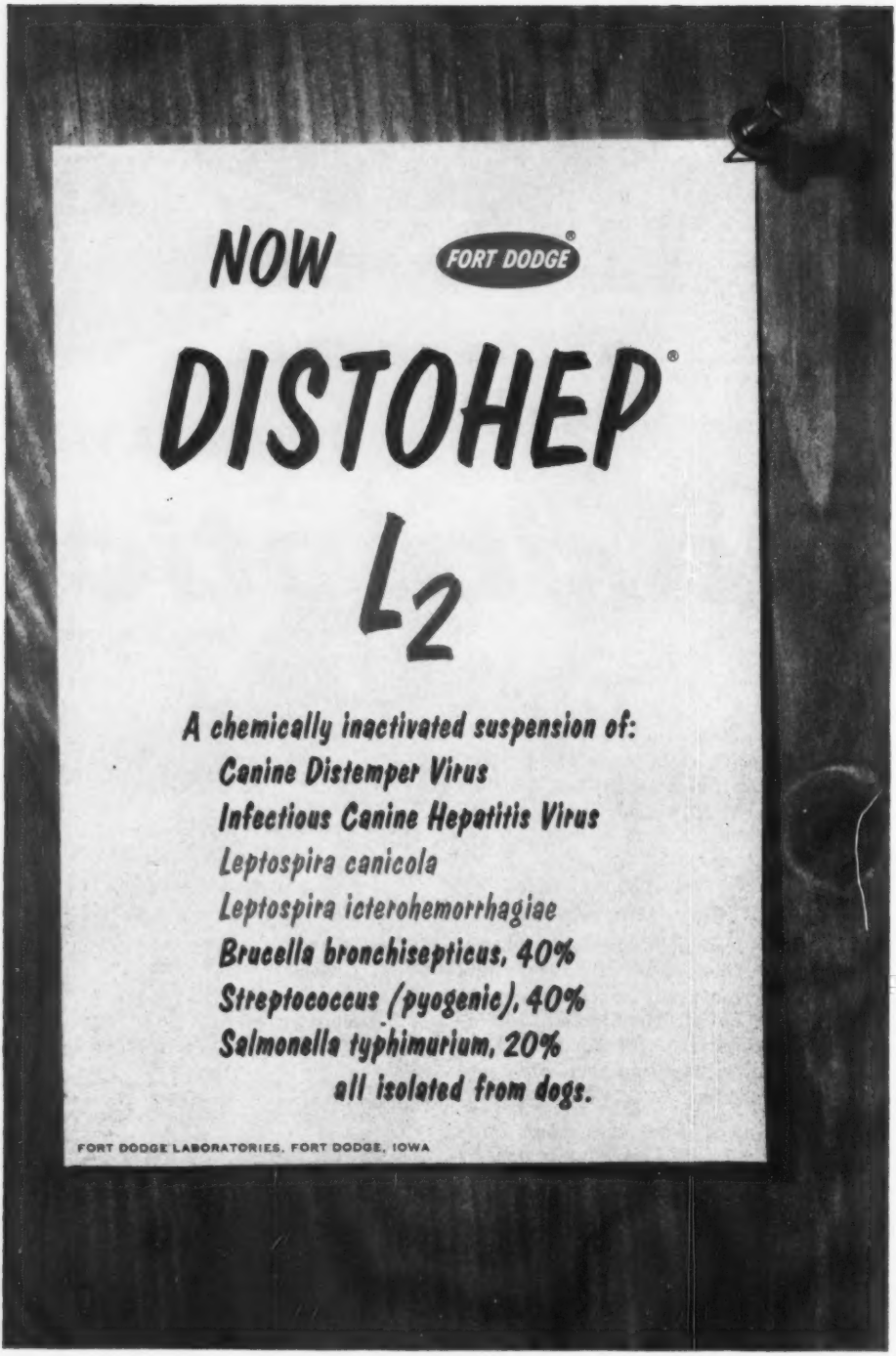
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November 15, 1961

Journal

OF THE
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Fibrotic Myopathy and Ossifying Myopathy in the Hindlegs of Horses

O. R. Adams, D.V.M., M.S.

FIBROTIC MYOPATHY in the hindlegs of horses results from a healed injury to the semitendinosus, semimembranosus, and biceps femoris muscles. The fibrotic lesion that occurs in the semitendinosus muscle is the most important because adhesions form between this muscle and the semimembranosus and biceps femoris muscles. These adhesions limit the action of the semitendinosus muscle causing an abnormal gait. This lameness most often occurs in Quarter Horses because of the type of work they perform.

Ossifying myopathy in the hindleg also results from previous injury to these muscles. Signs of lameness are the same as in fibrotic myopathy and are due to adhesions extending from a bony plate which forms over the semitendinosus muscle to the semimembranosus and biceps femoris muscles.

Thirteen cases of fibrotic myopathy and one case of ossifying myopathy in the hindleg have been diagnosed at the Colorado State University Veterinary Clinic since 1955. One case of ossifying myopathy has been observed in the foreleg.*

These lesions were first described by the author as myositis fibrosa and myositis ossificans.[1] This nomenclature was adopted after a review of the literature on the disease in man.[2] After a histologic study of the diseased musculature, the terms were changed to fibrotic myopathy and ossifying myopathy, since signs of inflammation were absent in the affected tissue.**

Etiology

Trauma is thought to be the cause of fibrotic myopathy.[1,2] Ossifying myopathy is a complication of fibrotic myopathy and is also thought to be caused by trauma. The fibers of the semitendinosus muscle may be injured when sliding stops are made in roping calves, or in other ways, such as resisting a sideline or catching a foot in a halter. Although both myopathic conditions are usually unilateral, one case of bilateral fibrotic myopathy was observed following a trailer accident. In some cases, the exact cause of the injury is not known. During the stage of myositis, no signs are present. When the injury heals, adhesions form between the involved muscles, and these adhesions cause the signs of lameness.

Fibrotic myopathy may result in ossification in the region of the affected muscle

From the College of Veterinary Medicine, Colorado State University, Fort Collins.

* Dr. W. F. Riley, Michigan State University, East Lansing: Personal communication, 1957.

** Pathologic studies were conducted by Dr. Rue Jensen, College of Veterinary Medicine, Colorado State University.

belly. The ossification is presumed to be caused by osteoblasts that have developed from fibroblasts by metaplasia. The condition is then termed ossifying myopathy.

Clinical Signs

The signs are due to adhesions between the semitendinosus muscle and the semimembranosus muscle medially, and between the semitendinosus and the biceps femoris muscle laterally (Fig. 1). These adhesions partially inhibit normal action of these muscles. In the anterior phase of the stride, the foot of the affected hindleg is suddenly pulled posteriorly for 3 to 5 inches, just prior to the time that it contacts the ground (Fig. 2). Usually, the lameness is most noticeable when the horse walks. The anterior phase of the stride is shortened, and consequently the posterior phase is lengthened. This abnormal gait is easily identified and may result from either fibrotic or ossifying myopathy. An area of firmness can be palpated over the affected muscles on the posterior surface of the affected limb at the level of the stifle joint and immediately above.

In 1 horse with ossifying myopathy, the muscle belly of the semitendinosus muscle was covered with a bony plate 10 inches long (Fig. 3). Bone tissue removed from this horse had normal bone structure.

Microscopic lesions of fibrotic myopathy consist of hyalinization of the muscle cell cytoplasm with loss of striations, and moderately pyknotic nuclei. There is no evidence of inflammation or neoplasia.

Diagnosis

Diagnosis is based on presence of the typically altered gait and is made by palpation of a hardened area on the posterior surface of the leg at the level of the stifle joint. In making a diagnosis, stringhalt should be considered. In stringhalt, the limb is pulled toward the abdomen. In fibrotic myopathy, the limb is pulled toward the ground in a posterior direction just prior to the time the foot contacts the ground. The limb is limited in the anterior phase of the stride by the adhesions formed and by the lack of elasticity in the affected area of the muscle belly. The limb is pulled posteriorly before the full length of stride can be reached.

Therapy

The most effective therapy involves surgical section or removal of a portion of the semitendinosus tendon at the level of the stifle joint. This may be done as a simple section of the tendon with a bistoury or by a more extensive operation, in which approximately 3 inches of the involved semitendinosus tendon is removed at the junction with the muscle belly before the tendon divides. The latter operation also includes separation of adhesions between the semitendinosus muscle and the

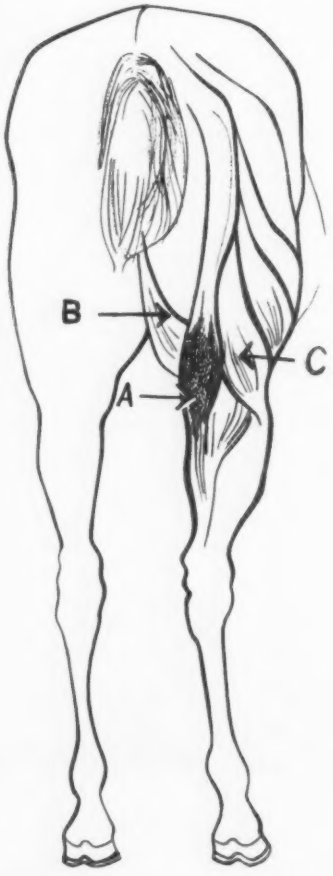

Fig. 1—The dark portion in the drawing represents an area of fibrotic or ossifying myopathy in the hindleg of a horse. A—semitendinosus muscle, B—semimembranosus muscle, and C—biceps femoris muscle.

semimembranosus and biceps femoris muscles.

For simple section of the tendon, the surgical area over the semitendinosus tendon at the level of the proximal end of the tibia is clipped, shaved, and prepared for surgery. Local infiltration of the affected tissues with 2% lidocaine hydrochloride* is used. The horse may be tranquilized if necessary. The operation may be done with the horse in either a recumbent or standing position. A small vertical incision is made over the semitendinosus tendon on the posterior side of the leg where it is easily palpated. A bistoury is inserted under the semitendinosus tendon, and the tendon cut near the junction with its muscle belly and before the tendon divides. The tendon is thick and digital palpation in the wound must be used to make sure that the entire tendon is cut. In some horses, new adhesions form following transection of the tendon, and subsequently 3 to 4 inches of the affected muscle tendon must be removed.

When a portion of the tendon is to be removed, the horse is tranquilized, cast with the affected side up, and given enough chloral hydrate to produce deep narcosis. The posterior aspect of the leg over the involved muscles at the level of the stifle joint and above is prepared for surgery and locally infiltrated with lidocaine hydrochloride as previously described. The affected limb should be extended to facilitate surgery. A vertical incision 6 inches long is made on the posterior aspect of the semitendinosus tendon. The tendon is identified and the adhesions to it from the semimembranosus and the biceps femoris muscles are removed. The muscle belly of the semitendinosus muscle is isolated, and a 3-inch portion is removed. The part removed should be comprised of 1½ inches of tendon and 1½ inches of muscle belly. Following removal, the remaining muscle immediately contracts and a large cavity results. The fascia is closed over the cavity with No. 2 catgut. The skin is then sutured with vertical mattress sutures of 0.6-mm. synthetic suture.** The wound is then protected with collodion and cotton. Tetanus antitoxin should be administered. Antibiotics usually

*Xylocaine, Astra Laboratories, Worcester, Mass.
**Vetafil, Norden Laboratories, Lincoln, Neb.

Fig. 2—The dotted lines indicate the action of the limb as it is pulled posteriorly instead of being put down in a normal fashion.

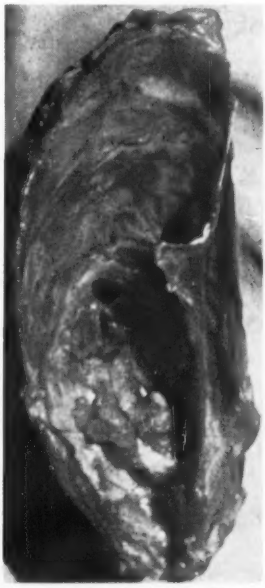

Fig. 3—A bony plate 10 inches long which was removed from the posterior surface of the semitendinosus muscle of a horse with ossifying myopathy. The view is of the inside concave surface and shows how the bony plate conformed to the shape of the semitendinosus muscle. The tendon of the muscle was at the bottom of the figure.

are not necessary, provided asepsis has been maintained during surgery.

When ossifying myopathy is present, the presurgical treatment is the same. After the incision, the skin is reflected and the bony cover over the semitendinosus muscle is dissected away. The adhesions on the medial and lateral sides of this muscle are cut, and the skin is then closed as described above.

Results

Some relief is evident in all cases. In most horses, removal of a 3-inch section of muscle will give better results than will simple section of the tendon. After healing, some horses will develop characteristic but less pronounced signs, but limb function is nearly normal and signs are not noticeable except at the walk. Occasionally, it will take 3 to 7 days for maximum benefits of surgery to be recognized.

Conclusion and Summary

Fibrotic myopathy and ossifying myopathy of the semitendinosus muscle in horses cause a typically abnormal gait that is easily identified. Diagnosis is based on observation of the typical gait and by palpation of the fibrosed or ossified area over the semitendinosus muscle at the level of the stifle joint. Normal use of the horse may be regained by surgical correction. In those horses in which signs persist after surgery, the gait is abnormal only when the horse walks.

References

[1]Adams, O. R.: Veterinary Notes on Lameness and Shoeing in Horses. Published by the author, Fort Collins, Colo., 1957.

[2]Adams, Raymond D., Brown, D. D., and Pearson, C. M.: Diseases of Muscle. Harper and Brothers, New York, N.Y., 1953.

Mink Toxoplasmosis

Toxoplasmosis was diagnosed in mink by serologic and histologic examination and confirmed by isolation of organisms in mice. Mortality was confined to kits. Losses in the kit crop were as high as 50% and were due mainly to neonatal deaths and abortion of dead kits at term.

On three of four affected mink ranches, ground muskrats, rabbits, and hogs, both fresh and frozen, had been fed during or just previous to the time losses occurred. On the fourth ranch, ground muskrats had been fed four years previously.

Organisms indistinguishable from *Toxoplasma gondii* were isolated from apparently healthy muskrats in the same areas, suggesting that they were a possible source of *Toxoplasma* infection in mink.—*Canad. J. Pub. Health, 52, (Sept., 1961): 392.*

Some Aspects of Foot Rot in Sheep

Douglas F. Watson, V.M.D.

OVER 20 YEARS have passed since the causative agents of foot rot in sheep were first described[1] and some principles for eradication were laid down. However, foot rot in sheep continues to be a problem. It has been suggested that if foot rot is not controlled, the sheep business will no longer exist as an economic factor. Some feel that there is no effective treatment and that, once a farm is infected, the causative agent always remains on the premises. Although it may be true that our methods of control are not perfect, they are effective. Perhaps the methods of control are not well enough known.

Over the past 15 years, I have been closely associated with sheep husbandry. When foot rot has been a problem, it has always been possible to eradicate the disease by utilizing the methods described here.

Causes of Foot Rot

Foot rot in sheep is caused by *Fusiformis nodosus*. This organism is always associated with another organism, *Treponema penortha* (*Spirochaeta penortha*). The disease is contagious and is characterized by separation of the horn from the underlying soft tissue, due to infection under the horn. The organisms appear to be obligatory parasites, and their habitat is the foot of sheep. Once the initial infection has become established other organisms invade. Even under the most favorable conditions, the organisms cannot live away from the host for more than two weeks.

Since the bacteria are present in the feet of infected or carrier sheep, infected material is deposited on the ground and then picked up on the feet of susceptible sheep. A healthy hoof may become infected if there is a break in the skin or horn of the foot. These breaks may result from wet grass, chafing, wounds, and most commonly from uneven growth of the hoof. Larvae of *Strongyloides sp.*, and *Bunostomum sp.* may provide entry for the organism.

Methods of Treatment

Foot rot can affect sheep only if the organism is in the soil, if it is being carried by chronically affected animals, or if it is introduced by purchase of infected or carrier sheep. Although unlikely, the organism may be spread by boots, car tires, or farm animals other than sheep.

Eradication is based on the fact that the organism cannot survive off the foot of the sheep for more than two weeks; therefore, any pasture that has not been grazed by sheep for more than two weeks may be considered free of the disease organism.

The first step in eradication is to examine the entire flock to determine if there are any signs of foot rot. The feet of all sheep are examined. All overgrown and diseased horn is trimmed away. A thorough search for pockets of infection is made. The use of a foot rot shears is recommended. Following trimming of the feet, the healthy sheep are put through a foot bath containing a suitable disinfectant as later described. These sheep are then confined on clean pasture, one on which no sheep have been for at least two weeks.

All affected sheep are subjected to a more thorough trimming of the feet and also given a foot bath. They are then placed in a special lot and treated individually and given foot baths at three-day intervals until complete recovery takes place. When they appear recovered, they are placed in a so-called "convalescent lot," if possible, before being returned to the clean flock.

From the Department of Veterinary Science, Virginia Polytechnic Institute, Virginia Agricultural Experiment Station, Blacksburg.

[1]Beveridge, W. I. B.: Foot Rot in Sheep. Parts 1, 2, and 3. J. Council Sci. & Indust. Res. Australia, 11, 1938): 1-20 (Abstr. in Vet. Bull., 9, (1939): 331-332).

Complete recovery can be assumed when the sheep no longer have lesions at weekly examinations for three weeks to a month.

Individual treatment may include antibiotics, 30% copper sulfate solution or 1 part of powdered copper sulfate to 2 parts of tar, or surgical removal of infected claws. Some sheep may be so badly affected that removal from the flock is advisable.

By this method, foot rot can be eliminated. It is not easy; sheep that have been returned to the clean flock may again develop foot rot because a carrier was not detected. Then it is necessary to start all over again.

The most satisfactory way to treat sheep is the use of a foot bath. Regardless of type of foot bath used, the feet should be trimmed properly and thoroughly cleansed before bathing. There should be enough disinfectant in the bath to cover the feet. One must make sure that the sheep stands in the solution.

For the foot bath, we have used 10% copper sulfate in water, 3 to 5% formalin in water, or more concentrated solutions with as much as 2 lb. of copper sulfate per gallon of water or 5 gal. of formalin per 100 gal. of water. At the more dilute level, the sheep must stand in the solution for five minutes. At the concentrated levels the time can be reduced to one minute per sheep. Although there are other useful drugs, these two are efficient and by far the most economical.

All new sheep that are destined to be incorporated into the main flock are treated as infected sheep and are subjected to thorough trimming, disinfection, and isolation for a month before being permitted to join the main flock. The same procedure is applied to any sheep taken off the farm for any purpose and then returned.

Summary

The cause of foot rot in sheep is *Fusiformis nodosis*. Control may be effected by trimming feet and treating with therapeutic foot baths. An eradication plan incorporated the following procedures: establishment of (1) a clean flock of sheep with no apparent disease; (2) a convalescent flock into which sheep, apparently recovered after treatment, were held for two to three weeks; and (3) an infected flock with sheep under intensive treatment.

Trichinosis Reduced by Garbage Cooking

Examination of diaphragms from 789 swine originating from licensed garbage-feeding premises in four Canadian provinces revealed the presence of trichinellid larvae in only two diaphragams (0.25%), both from the same establishment. This is considerably lower than the 6 to 11% reported for garbage-fed swine in the United States during the years 1933 to 1939, during which time there were few garbage-cooking restrictions. It was suggested that the swine on the infected premises acquired trichinellid infection by either faulty management or by eating infected rats.—*Canad. J. Pub. Health, 52, (Aug., 1961): 339.*

Clinical Coccidiosis in Wisconsin Cattle

D. L. Peardon, M.S.; M. R. Hasche, M.S.; A. C. Todd, Ph.D.; R. E. Hall, D.V.M.

PARASITOLOGISTS invariably list coccidiosis in their estimates of important diseases of cattle. Some investigators[4] have concluded that economic loss in the United States from bovine coccidiosis must be nearly as great as the losses due to coccidiosis in chickens. According to an estimate[6] made in 1949, bovine coccidiosis constituted the third most important of all cattle diseases.

In the last two decades, when investigators were studying the economics of bovine coccidiosis in the South, clinical reports about coccidiosis in cattle in the North almost disappeared. Bovine coccidiosis became a southern disease by reputation despite an earlier and extensive series of reports concerning the disease in the northern states of North Dakota, Kansas, Montana, Nebraska, Washington, Pennsylvania, and New York.[1,5,7,10-16]

The prevalence of bovine coccidiosis presently is being investigated in Wisconsin. Ten species of *Eimeria* have been found in cattle in the state. Infections have been found in each Wisconsin county.[8]

Observations on six cases of clinical bovine coccidiosis in Wisconsin are reported here.

Case Reports

Case 1.—In early May, young calves in a purebred Brown Swiss herd became depressed, developed scours, and "went down." The clinical signs were described to the local veterinarian who advised treatment with antibiotics. When the signs persisted, the veterinarian initiated an added course of treatment with astringents. Later, when bloody scours developed, the veterinarian made a tentative diagnosis of coccidiosis which was not confirmed because, on microscopic examination of bloody feces from the calves, large numbers of oocysts were not found.

The disease was confined to calves 4 to 12 weeks of age. The herd history revealed that a 10-month-old show calf had the same signs the previous fall, shortly after its return from a dairy show. Later in the year, 1 calf, 3 months old, developed severe, bloody scours but recovered after supportive treatment. A remodeling of the calf barn which precluded practice of some regular sanitary procedures preceded the infections. A number of calves passed large amounts of fresh blood in their feces prior to marked production of oocysts.

Fecal samples were collected from the group of calves which ranged in age from 4 weeks to 5 months. The total number of oocysts in a 5-Gm. sample ranged from 0 to 8,016. Nine species of *Eimeria* were found in which three species, *E. zurnii*, *E. bovis*, and *E. ellipsoidalis*, predominated.

Case 2.—In late October, coccidiosis occurred in Hereford calves at the University of Wisconsin. Clinical signs were observed in a 6-week-old calf. A direct smear examination made of a fecal sample collected from this calf established the presence of large numbers of *E. bovis*, *E. zurnii*, and *E. ellipsoidalis* oocysts. A total of 52,880 oocysts were present in a 5-Gm. sample.

Fecal samples in series were collected from the entire herd which consisted of 35 calves of both sexes, ranging in age from 1 to 17 months; 14 cows, 4 years old; 11 cows, 5 years old; and 1 bull, 5 years old. A total of 166 samples was examined.

The number of oocysts per 10-Gm. sample ranged from 0 to 5,610. About 10% of the samples were negative. The number of species per sample ranged from one to nine. Ten species were present in the herd.

Case 3.—Clinical coccidiosis occurred in late April in a herd of 220 Aberdeen Angus heifers weighing approximately 700 lb. each. The animals had been put in a feedlot as 400-lb. calves in early October.

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Bloody feces had been observed in a few in the latter part of February. An antibiotic was then fed under veterinary direction. The bloody scours continued. Upon consultation a definitive diagnosis of coccidiosis was made.

A total of 10,504 oocysts per gram of feces was found in 1 heifer. Approximately 97% of the oocysts were *E. zurnii.* No evidence of acid-fast bacteria, *Vibrio,* or *Salmonella* was found upon laboratory examination.

About 25 heifers had clinical signs in late April and were isolated. No deaths occurred. In 16 fecal samples taken at random, the numbers of oocysts per 10-Gm. sample ranged from 52 to 29,588. The number of species in an individual sample ranged from two to eight. Of the nine species present, *E. bovis, E. zurnii,* and *E. ellipsoidalis* were predominant.

Case 4.—Bloody scours developed in an 11-month-old Brown Swiss steer in late August at the Wisconsin Central Animal Disease Diagnostic Laboratory. The steer strained to defecate, had a dull coat, and its tail and hindquarters were soiled with feces. There were no signs of impeded locomotion, anorexia, or nervousness. Coccidiosis was suspected and a fecal sample was taken.

The animal had been held in a paddock near the laboratory and served as a blood donor. It shared the paddock with several sheep, but it had been isolated for four months from other cattle.

Large numbers of oocysts were found upon microscopic examination. Six species of *Eimeria* were present. Of the 172,532 oocysts in a 5-Gm. fecal sample, 84% were *E. zurnii.*

Case 5.—In early November, 4 of 9 Holstein-Friesian calves, ranging from 2 to 10 months of age, died within five days on a farm in southwestern Wisconsin. Bovine coccidiosis was diagnosed tentatively since the animals had bloody diarrhea, emaciation, and weakness. A fecal sample was collected and examined.

In the two preceding years, the replacement cattle in the herd had had scours after a few weeks in winter quarters after summer pasturing. Coccidiosis had not been diagnosed previously.

A 5-Gm. fecal sample was examined and 90,844 oocysts were counted; 72.5% of these were identified as *E. zurnii.* Five other species of oocysts also were observed.

Case 6.—In November, clinical coccidiosis developed suddenly in 2 of 7 Guernsey heifers on a farm in north central Wisconsin. The heifers were 8 months to 2 years old. A presumptive diagnosis was made by the attending veterinarian, and the diagnosis was confirmed by a laboratory.

A 5-Gm. fecal sample from 1 heifer contained 152,986 oocysts. Of these oocysts, 68% were identified as *E. zurnii;* the majority of the remainder, as *E. subspherica.* Seven species of coccidia were involved.

Discussion

The observations reported here imply that coccidiosis may be a widespread and serious problem in cattle in northern climates. The work on bovine coccidiosis at this laboratory was initiated after a series of discussions in local and state veterinary associations in the North in which problems with clinical coccidiosis were reported by many practitioners. Most of the emphasis was placed on coccidiosis in feedlot cattle.

Ten species of bovine coccidia are known to be prevalent in Wisconsin. Study of these field occurrences indicates the most pathogenic species are *E. zurnii, E. bovis,* and *E. ellipsoidalis. Eimeria zurnii* predominated in each occurrence.

According to Alabama investigators, coccidiosis was most often seen after the change from summer pasture to the confinement of winter quarters.[4] The disease in Wisconsin follows such a pattern, but it also seems to be related to overcrowded conditions, resulting in part from growth of the animals themselves, which precipitates rapid re-exposure as numbers of oocysts accumulate. Although many northern dairy farmers use an age-segregation system for dairy calves, manure removal from the pens is often neglected as the winter advances. The portable pen system for dairy calves is not a widespread management practice in this climate, not even in the summers.[3]

In two instances in Wisconsin, calves were housed through the winter or confined in a feedlot, and they developed coccidiosis as late as April and May. Three other times coccidiosis occurred in housed dairy calves in the fall. In 1 of these calves, coccidiosis developed as early as October and, in the other 2, in November. In the Aberdeen

Angus heifers on full feed, first signs of clinical coccidiosis did not appear until five months after they entered the feedlot. The two occurrences in spring may have been correlated with the seasonal variation of temperature characteristic of Wisconsin weather.

The aforementioned cases indicate again that the breeds of the cattle are not a factor in clinical coccidiosis. The age range of the animals involved was somewhat wider than usually is reported. The youngest calf clinically affected was less than 4 weeks old. On the other hand, Aberdeen Angus heifers on full feed and weighing 700 lb. also were susceptible, and the disease was clinically apparent. The 11-month-old Brown Swiss steer either came in contact with an accumulation of newly infective oocysts or developed clinical coccidiosis when its carrier state was transformed into an acute infection because of the stress of handling or loss of blood as a donor.

The cases in Wisconsin cattle reported here were not characterized by high mortality rates, at least during the period of our observations. We believe that the feeding programs furnished northern cattle aid greatly in resistance to coccidiosis. Owners of the cattle involved were not conscious of economic loss except in the instances of the owner who lost 4 of 9 Holstein-Friesian calves and the owner of the Aberdeen Angus heifers who was convinced that he had sustained damaging loss of time in marketing his animals.

The lack of consistent and direct diagnosis of bovine coccidiosis in the North appears to be due to several factors. In those instances in which the consulting or attending veterinarian was a member of this department or of the Wisconsin Central Animal Disease Diagnostic Laboratory, a microscope was readily available for direct examination of manure samples. In the instances in which the disease was first reported by the owners to a veterinarian via telephone, the reports of scours and pneumonia led directly to the employment of antibiotics either by injection or in feed. We have not found antibiotics to be effective as coccidiostats in cattle.

In two instances, a presumptive diagnosis of coccidiosis was arrived at after antibiotics had failed to arrest the disease. In one instance, microscopic examination for oocysts by the veterinarian was unsuccessful even when a bloody fecal sample was examined, apparently because the infection was still in the stage of second generation schizogony.

In six cases, coccidiosis progressed until the infections subsided either from limitation by the natural cycle of the parasite[2] or from development of immunity within the herd, or from development of resistance afforded by supportive treatment. In our opinion, none of the present bovine coccidiostats can arrest a clinical case of the disease.

Summary and Conclusion

Six instances of bovine coccidiosis are reported from cattle in Wisconsin. The disease evidently is more widespread and more serious in cattle than was formerly suspected in this state.

Although ten species of bovine coccidia have been found in Wisconsin, *Eimeria zurnii, E. bovis,* and *E. ellipsoidalis* appear to be most pathogenic; *E. zurnii* was the predominant species in all six cases reported. Five of the six cases occurred during the period when cattle were confined in winter quarters. Three cases occurred during the fall months and two cases as late as April and May. The breed of cattle was not a factor in clinical coccidiosis.

There was a wider range of ages in the cattle affected than was normally expected. Cases occurred in 4-week-old dairy calves and in yearling Aberdeen Angus heifers in a feedlot. The most severely affected cattle ranged from 6 weeks to 8 months old.

A direct and accurate method of diagnosing bovine coccidiosis in the field and a useful coccidiostat are needed.

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Scrotal Hernia in a Dog

R. L. Leighton, V.M.D.; J. T. Cordell, V.M.D.; Bruce H. Ewald, D.V.M.

APPARENTLY, INGUINAL and scrotal hernia in the male dog is uncommon.[3] A search of the literature did not reveal any cases reported in English language publications. Two cases of incarcerated inguinal hernia in male dogs have been reported from the surgical clinic of the Royal Veterinary College in Stockholm.[2] Two reports were found in the German literature.[4,5] In one, three cases of inguinal hernia and 11 cases of scrotal hernia in male dogs are listed.[4]

One report on the anatomy of the inguinal canal of the dog concludes that the difference in shape and especially the posterior conformation renders the bitch more vulnurable to hernia than the dog.[1]

In view of the apparent rarity of inguinal and scrotal hernia in the male dog, the following case is reported.

Case Report

A 1-year-old Basset Hound was referred to the surgical clinic Jan. 3, 1961, by a local veterinarian for surgical treatment of a marked swelling of the right scrotum which extended cordlike to the inguinal ring. The surrounding area was edematous; there was evidence of pain when touched; and the right testicle seemed several times normal size. The swelling first occurred ten days previously, receded somewhat a day later, then became enlarged again. The dog was hospitalized for surgical exploration.

Under pentobarbital sodium anesthesia, an incision was made over the distended cord at the inguinal ring. The tunica vaginalis was incised, revealing a large, tightly packed, reddish mass of omentum which extended into the scrotum. Pressure upon the testicular artery and vein seemed responsible for the edema.

The omentum was unaffected in the abdomen, but the herniated portion was so markedly inflamed that it was amputated. The enlarged inguinal canal was closed with interrupted 0 chromic gastrointestinal catgut. The testicle was not removed. The tunica vaginalis was sutured with interrupted 000 gastrointestinal chromic catgut. The subcutaneous tissues were closed with 00 general closure chromic catgut, and the skin was closed with 00 interrupted braided stainless steel sutures.

The edema subsided in a few days, and the dog was discharged five days after surgery.

Antibiotics were given intramuscularly for five days. A protective dose of canine distemper serum antibody concentrate was given.

Head, surgical service (Leighton), staff veterinarian (Cordell), and former staff veterinarian (Ewald), Animal Medical Center, New York, N.Y. Dr. Ewald is now in practice in Eastchester, N.Y.

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Unilateral Renal Agenesis with Ipsilateral Cryptorchidism and Perineal Hypospadias in a Dog

L. Z. McFarland, D.V.M., Ph.D., and
E. Deniz, Dr. med. vet.

AGENESIS of the kidney is uncommon in the dog, having a frequency of about 1 in 400 according to one investigator[4] who observed 3 cases in 1,250 dogs. Absence of either the right kidney[3] or the left kidney[1,2,5,6] has been reported in the dog.

A case of renal agenesis with concurrent malformations of the urogenital system in an 8-month-old, male Beagle is reported here.

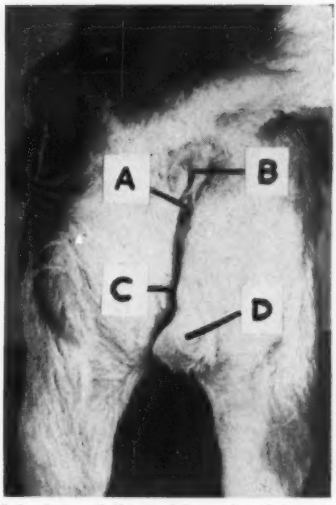

Fig. 1—Caudal view of dog with perineal hypospadias. The urethral orifice (A) is located below the anus (B). A urethral groove (C) extends forward along the midline, and only the right half of the scrotum (D) containing the descended testis is developed.

Anatomical Findings

A condition of perineal hypospadias existed with the urethral orifice situated about 1 cm. below the anus (Fig. 1). A pinkish colored urethral groove (Fig. 1) extended from this orifice forward along the midline to the glans penis. The prepuce had not united ventrally (Fig. 2); the glans penis was underdeveloped; and the penile urethra and corpus cavernosum urethra were absent.

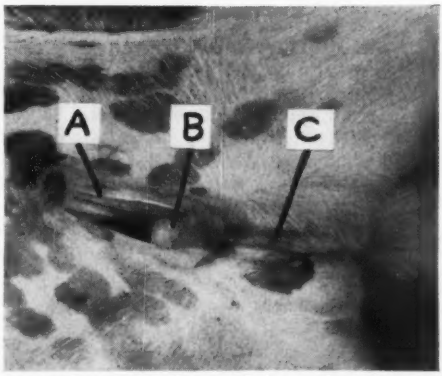

Fig. 2—In a ventral view of the urethra, the cleft prepuce (A), the underdeveloped glans penis (B), and the urethral groove (C) are visible.

Only the right testis and associated structures had descended through the inguinal canal and entered the right half of the scrotum. The left half of the scrotum was not represented (Fig. 1). The left testis and associated structures were retained in the abdominal cavity lying opposite the right kidney (Fig. 3), with the left testis remaining about one fourth as large as the right. The inguinal ligament of the testis was attached to the ventral body wall near the inguinal canal instead of passing through it.

The left kidney and its vascular supply were absent (Fig. 3), and only a small imperforate ureter, which terminated typically at the bladder, persisted. The right kidney was large (ca. 0.7% body weight) but not abnormally hypertrophied.

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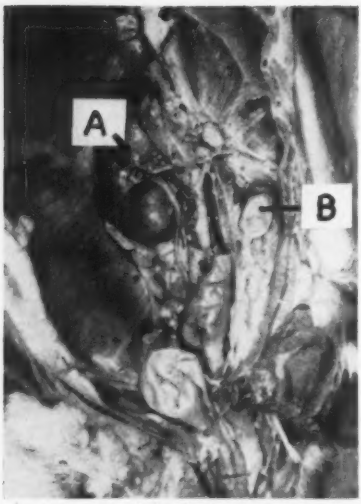

Fig. 3—View of abdominal cavity showing the right kidney (A) and the retained left testis (B). The left kidney and associated vessels are absent.

Discussion

The kidney and ureter are derived from the embryonic metanephros and metanephric tubules, respectively, whereas the testis and epididymis arise from the mesonephros and mesonephric tubules, respectively. The congenital defect observed in this dog indicates a developmental failure of the metanephros independent of the developing metanephric tubules or mesonephros, as evidenced by the persistence of a ureter and the retained testis.

It appears that renal agenesis is not a hereditary defect, but rather an individual developmental deviation induced by one of several factors, such as altered *in utero* environment, hypoxia, avitaminosis, and defective ovum.

In the dog, congenital absence of the kidney affects the male more often than the female and involves the left kidney more frequently than the right. Depending on the physiologic state of the dog, the remaining kidney may be either normal in size or hypertrophied.

Since renal agenesis was associated with cryptorchidism and perineal hypospadias in this case, it is probable that a common factor affected the urogenital system of this dog during its early developmental period.

Summary

Congenital absence of the left kidney was observed in an 8-month-old male Beagle. Other defects of the urogenital system included retention of the left testis and perineal hypospadias.

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Dermoid Cysts on Boxers

According to a report in a Swiss journal, dermoid cysts occur commonly on the top of the skull in Boxers. Cysts look like tumors or like an elongated scar without hair but with normal pigmentation. Well-developed cysts may fluctuate on palpation. Some are resorbed and disappear. They are believed to be heterotopic and of hereditary origin. The cysts, harmful only from the aesthetic point of view, can be removed surgically. Affected animals should not be used for breeding.—*Schweiz. Arch. Tierheilk., 6, (June, 1961): 308.*

Disophenol, an Injectable Anthelmintic for Canine Hookworms

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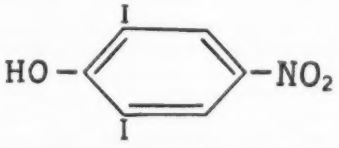
DISOPHENOL* is an injectable anthelmintic agent for treatment of dogs infected with *Ancylostoma caninum*, the common dog hookworm, *Ancylostoma braziliense*, the southern dog hookworm, and *Uncinaria stenocephala*, the fox hookworm. Disophenol is the generic designation for 2,6-diiodo-4-nitrophenol which has the following structural formula:



This compound was selected from many analogues because of its efficacy against *A. caninum* in dogs.[6]

Laboratory results on the efficacy and the safety of disophenol by oral and parenteral routes of administration are presented in this paper.

Materials and Methods

Eighty-three, 8- to 16-week-old, mixed-breed dogs, free from natural infections of hookworms, were used for the efficacy studies. They were experimentally infected, *per os*, with single doses of 150 to 300 infective larvae of *A. caninum* or *A. braziliense* or 75 to 100 infective larvae of *U. stenocephala*. Two dogs naturally infected with the latter species were also treated.

Dogs infected with adult worms were treated 28 or more days after larval inoculation when the eggs per gram of feces (e.p.g.) was relatively constant from day to day. The effects of disophenol against the immature parasitic stages of *A. caninum* and *A. braziliense* were also studied in 25 of the aforementioned dogs.

The dogs were treated about two hours after they had been fed. Oral doses of disophenol were given as a dry powder in gelatin capsules; for parenteral doses, the powder was usually dissolved in 1 or 2 cc. of various parenteral formulations. These are not differentiated in the results since their activities and toxicities were similar to those of the 1:1 water and polyethylene glycol solvent which was finally chosen. Sodium hydroxide was used to increase solubility in the aqueous formulations.

Fecal egg counts were made by the Stoll technique one day before or on the day of treatment and on the third or ninth day after a parenteral or oral treatment, respectively. At necropsy one day later, the gastrointestinal tract was removed and examined thoroughly for worms. The viscera and the injection sites were examined grossly for lesions.

The anthelmintic effect was determined by either of two methods: (1) the standard critical test which compared the number of worms eliminated in the feces after treatment to those retained in the dog at necropsy or, more frequently, (2) a comparison of the pretreatment e.p.g. and the number of worms at necropsy.

With *A. caninum* infections, the following formula was used:

$$\text{Efficacy (\%)} = \frac{\text{Pretreatment e.p.g.} - (50 \times \text{worms at necropsy})}{\text{Pretreatment e.p.g.}} \times 100$$

The conversion factor of 50 in the preceding formula was derived from data indicating that under our experimental conditions, on the average, approximately 50 e.p.g. (46.5 ± 7.7 standard error) was nearly equivalent to one adult worm in the canine host. These data were obtained from 80 experimentally infected, untreated dogs of comparable weight,

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The synthesis of disophenol and its analogs by E. Benz, L. Goldman, A. E. Lanzilotti, J. K. Paul, and L. N. Starker of the Organic Chemistry Research Section, Lederle Laboratories, and the technical biological assistance of Joseph Entwistle are gratefully acknowledged.

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age, and previous exposure to hookworm infection as the dogs in the efficacy studies. The dogs were given a similar number of infective larvae and were fed the same commercial canned dog feed in similar quantities. The egg counts on these untreated animals were made at the same and various other times with respect to the course of the infection as in the treated dogs. In this study, the mean per cent efficacy (Table 1) for each dose regimen was derived by averaging the individual values for each dog.

In tests against the parasitic larval stages, the criterion of activity was a marked reduction in the e.p.g. counts of treated dogs compared with their untreated controls. These counts were made between 13 and 23 days after larval inoculation.

The effects of disophenol were observed on natural infections with the ascarids, *Toxocara canis and Toxascaris leonina;* the whipworm, *Trichuris vulpis;* and the tapeworm, *Dipylidium caninum;* when these were present in the dogs experimentally infected with hookworms.

To determine its margin of safety, the effects of various doses of disophenol were observed in an additional 86 dogs not infected with hookworms. Most of these varied from six weeks to one year of age.

Results

Efficacy of Disophenol

Ancylostoma caninum.—The experimental infections averaged 89 adult hookworms with a standard error of 6 worms as estimated from the pretreatment e.p.g. values of 43 dogs (Table 1).

Disophenol (5.0 mg./kg.) was more than 96% effective when given either orally or intramuscularly. Of the 17 dogs treated, 6 had no worms, 7 had 1, and 4 dogs had 3, 4, 7, and 12 worms, respectively, at necropsy. Although 5.0 mg./kg. subcutaneously markedly reduced the e.p.g. values, 7.5 mg./kg. was necessary to achieve essentially complete worm removal. At necropsy, no hookworms were found in 8 of 11 dogs treated with the latter dose; the other 3, each had 1 worm. The mean pretreatment e.p.g. of these 11 dogs was 4,375, indicating an average of 88 worms. Critical tests on 2 of these dogs and on 4 with concurrent infections of *U. stenocephala* (Table 2), also indicated the high efficacy against *A. caninum* of 7.5 mg./kg., subcutaneously. Some *A. caninum* were passed within 24 hours after treatment, and all were eliminated within 48 hours.

Single doses of disophenol which were highly effective against adult hookworms were ineffective against the immature stages of *A. caninum.* No activity was present with treatment before 14 days postinoculation; partial elimination of adult worms occurred between 14 and 19 days.

Uncinaria stenocephala.—This species was not as effectively controlled as were the other hookworms by a subcutaneous dose of 7.5 mg./kg., but 10.0 mg./kg. gave complete elimination of adult worms in 4 dogs (Table 2). In all dogs given 10.0 mg./kg. and in 4 of 6 dogs given 7.5 mg./kg., egg counts of *Uncinaria* were reduced by 100%; in the remaining 2 treated with 7.5 mg./kg., egg counts were reduced by 98%.

Ancylostoma braziliense.—A 100% efficacy against adults of this species was obtained with subcutaneous injections of 7.5 mg./kg. in 6 dogs (Table 3). No worms were found at necropsy three and four days after treatment but, in test B with necropsy two days after treatment, 4 and 8 dead worms, respectively, were found in 2 dogs.

A dose of 5.0 mg./kg., given intramuscularly, was ineffective against 7-day-old parasitic larvae in 2 dogs.

Other Helminths.—There was no indication that disophenol was active against

TABLE 1—The Effects of Single Doses of Disophenol on Adults of *Ancylostoma caninum* in Dogs

Method of treatment	Dose (mg./kg.)	No. dogs	Pretreatment e.p.g. Mean	Pretreatment e.p.g. (s.e.)*	Egg reduction (%) Mean	Egg reduction (%) (s.e.)	No. worms at necropsy Mean	No. worms at necropsy (s.e.)	Efficacy (%) Mean	Efficacy (%) (s.e.)
Oral, capsule	2.5	4	4650	(300)	59.0	(17.0)	31.0	(18.0)	61.0	(21.0)
	5.0	11	4725	(800)	98.0	(1.0)	1.0	(0.4)	99.0	(0.4)
	10.0	1	6300	.....	100	.....	0	.....	100	.....
Intramuscular	2.5	2	4500	(100)	74.0	(4.0)	31.0	(10.0)	65.0	(12.0)
	5.0	6	5400	(970)	99.7	(0.3)	3.0	(2.0)	98.0	(1.0)
	7.5	2	3850	(50)	99.0	(1.0)	0	.....	100	.....
Subcutaneous	5.0	4	2850	(140)	97.0	(3.0)	21.0	(10.0)	63.0	(14.0)
	7.5	11	4375	(400)	99.8	(0.2)	0.3	(0.1)	99.6	(0.1)
	10.0	2	3400	(100)	100	.....	0	.....	100	.....

*s.e. = standard error.

TABLE 2—The Effects of Single Subcutaneous Injections of Disophenol on *Uncinaria stenocephala* and on Some Concurrent Infections of *Ancylostoma caninum* in Dogs*

Dose (mg./kg.)	U. stenocephala			A. caninum		
	No. of worms		Efficacy (%)	No. of worms		Efficacy (%)
	Eliminated	Retained		Eliminated	Retained	
7.5**	1	3	25	.....	.....	.....
7.5**	4	27	13	3	0	100
7.5	25	0	100	.....	.....	.....
7.5	77	9	90	102	0	100
7.5	18	3	86	16	0	100
7.5	8	10	44	43	0	100
10.0	29	0	100	.....	.....	100
10.0	98	0	100	124	0	100
10.0	43	0	100	113	0	100
10.0	14	0	100	52	0	100

*Each line represents data from 1 dog. **Natural infections; all others experimentally established.

concurrent infections of ascarids, trichurids, or *Dipylidium* in 16, 3, and 10 dogs, respectively, treated with parenteral doses which were highly effective against hookworms. No apparent activity was observed against ascarids in the three natural infections in the dogs given effective oral hookworm treatments.

None of these other helminths were observed in the feces after treatment, and all specimens recovered at necropsy were alive. For example, a dog treated with 7.5 mg./kg., subcutaneously, passed only hookworms in the feces, but 20 ascarids and 150 trichurids were recovered at necropsy four days later. Another dog did not pass trichurids during the first four days after treatment with a single intravenous injection of 7.5 mg./kg., nor was the trichurid egg count of 2,400 e.p.g. reduced after 13 days.

TABLE 3—The Effects of Single Subcutaneous Injections of 7.5 mg./kg. of Disophenol on *Ancylostoma braziliense* in Dogs

Test	No. dogs	Pretreatment e.p.g. (mean)	Egg reduction (%)	No. live worms at necropsy	Efficacy (%)
A	2 treated	2,600	100	0	100
B	2 treated	2,100	100	0	100
C	2 treated	4,000	100	0	100
	1 untreated	2,900	0	41	.....

Toxicity of Disophenol.—In acute toxicity studies (Table 4), the minimal lethal oral dose of disophenol was between 100 and 200 mg./kg. Emesis occurred within one to four hours after single oral doses in 2 of 12, 4 of 10, and 3 of 8 dogs given 10.0, 15.0, and 25.0 mg./kg., respectively; emesis was consistently produced after single oral doses of 50.0 mg./kg. and higher. The data suggest a twentyfold margin of safety between effective and maximum tolerated single oral doses, if the possible protective effect of emesis is discounted.

Single intramuscular and subcutaneous doses of 30.0 mg./kg. were safe in a total

TABLE 4—The Toxicity of Single Doses of Disophenol in Dogs

Method of administration	Doses (mg./kg.)											
	5.0	7.5	10.0	15.0	20.0	25.0	30.0	36.0	40.0	50.0	100.0	200.0
Oral	0*		0	0		0				0	0	2
	—	.....	—	—	.....	—	.....	.....	.....	—	—	—
	15**†		12‡	10‡		8‡				2‡	3‡	2‡
											—	
Intramuscular	0	0	0		0		0		0	4		
	—	—	—	.....	—	.....	—	.....	—	—	2	.....
	11†	2†	5		6		1		1	4‡	2‡	
Subcutaneous	0	0	0	0	0		0	1	2	3		
	—	—	—	—		.....	—	—	.....	—	—	.....
	4†	35†	6†	11§	1		10‖	1	2	3		

*Number of dead dogs. **Total number of dogs. †Infected with hookworms. ‡Emesis, see text. §Included 4 pups, 1 day old, and 4 pregnant females.[5] ‖Included 3 pups, 2 days old.[5]

of 11 dogs; doses of 36.0 mg./kg. and above were fatal, except in 1 dog given 40.0 mg./kg., intramuscularly. Emesis occurred after parenteral administration only at lethal intramuscular doses. The margin of safety for the recommended single subcutaneous dose of 7.5 mg./kg. was fourfold, and disophenol appeared equally safe for very young puppies, mature dogs, and for pregnant females.

Cumulative toxicosis may occur when disophenol is administered repeatedly. No toxicosis was observed when a single subcutaneous dose of 3.5 mg./lb. of body weight (7.7 mg./kg.), or twice this dose, was given once weekly for 8 weeks to 4 and 3 dogs, respectively, in gross toxicity studies. However, with repeated daily subcutaneous injections of 7.7 mg./kg. given to 4 dogs, 3 died six, seven, or eight days after treatment began, and only 1 dog survived the planned regimen of seven successive daily doses.[5] With repeated daily oral administration, deaths occurred in 1 dog after three and 1 dog after four doses of 25.0 mg./kg.; in 1 dog after five and in 3 dogs after 6 doses of 12.5 mg./kg.; and in 1 dog after 7 and in 1 dog after 16 doses of 9.0 mg./kg. Clinical signs in dogs given lethal doses were panting and increases in respiration, temperature, and heart rate.

Disophenol was tolerated relatively well in detailed toxicologic and pathologic studies on 2 young beagles given 5.0 mg./kg. orally once a day for 44 days.* Significant adverse findings were slight and variable increases in blood urea nitrogen (values did not exceed 26.0 mg./100 ml.) and a rapid onset of rigor mortis after euthanasia. A 60-day regimen of 5.0 mg./kg. orally once daily in 4 dogs, observed grossly by us, produced only slightly retarded weight gains in 3 dogs, and no effect in the fourth dog.

In some dogs given effective antihookworm doses, intramuscularly, there were signs of transitory pain, whereas in dogs given subcutaneous injections there were no overt reactions. Other than the usual tissue reactions to any injection, no gross pathologic signs were observed at any injection site of the dogs when examined at necropsy.

*Toxicologic and pathologic studies by Drs. J. A. Kaiser and W. Layton, respectively, and by their associates, Experimental Therapeutics Research Section, Lederle Laboratories Division, American Cyanamid Company.

Discussion

When given by parenteral injection, disophenol differs in efficacy from other compounds available for the treatment of canine hookworms.

Special pre-treatment and post-treatment measures such as withholding food or purging are unnecessary, and fully fed dogs may be treated. The parenteral activity of disophenol in low doses permits the use of a single formulation which is convenient for accurate and rapid treatment of dogs of any weight. The subcutaneous method of administration is preferred since the intramuscular route may produce some signs of transitory pain.

Disophenol has been formulated so that 0.1 cc. of parenteral solution per pound of body weight will contain the presently commercially recommended subcutaneous dose of 7.7 mg./kg. Doses of 7.5 mg./kg., by this route, removed an estimated 99.6% of *A. caninum* from 15 dogs and 100% of *A. braziliense* from 6 other dogs. At necropsy four days after a single injection, no hookworms were found in 18 of these 21 dogs, and the other 3 dogs each retained only 1 worm. Effects against *U. stenocephala* were variable with 7.5 mg./kg., but 10.0 mg./kg. was 100% effective in 4 dogs.

The mortality and morbidity caused by *A. caninum* and *A. braziliense* in dogs in the United States are well known, as is the public health significance of these parasites in cutaneous and possibly visceral larva migrans in man.[1] *Uncinaria stenocephala* has been found less frequently south of latitude 50 degrees North,[4] but a pooled 17% incidence has been reported from Illinois, Indiana, Kentucky, Michigan, Ohio, and Tennessee,[3] and recently this species has been observed in dogs from New York.[2] This hookworm is more common in Canada and Europe and has been reported from Australia.[4]

With disophenol, as with other available anti-hookworm agents, retreatment after two or more weeks may be necessary to eliminate reinfections or hookworms immature at the time of initial treatment. No toxicosis was observed when twice the recommended dose was given once a week for eight weeks, but deleterious effects may develop when repeated injections of the recommended dose are administered daily.

The fourfold margin of safety of single subcutaneous doses is considered adequate for a therapeutic agent which can be given

in precise amounts proportional to body weight by injection. Injection avoids variable toxicosis due to differences in absorption from the gastrointestinal tract.

Summary

Disophenol (2,6-diiodo-4-nitrophenol) was found to be an anti-hookworm agent effective against *Ancylostoma caninum* and *Ancylostoma braziliense* when given as a single subcutaneous dose of 7.5 mg./kg. to dogs which were not fasted. Efficacy was also obtained with similar oral and intramuscular doses. A subcutaneous dose of 10.0 mg./kg. appeared to be necessary for complete elimination of *Uncinaria stenocephala*, which is less common than *Ancylostoma spp.* in the United States. Activity against ascarids, trichurids, tapeworms, and immature hookworms was not observed.

No toxicosis in dogs has been observed with single parenteral doses of 7.5 or 10 mg./kg.; doses of 15.0 or 30.0 mg./kg. were also safe, whereas doses of 36.0 mg./kg. and higher were lethal.

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Parakeet Dander Pneumonitis

In persons with respiratory symptoms occurring after exposure to parakeets, the possibility of parakeet dander pneumonitis (acute psittaco-kerato-pneumonconiosis) should be considered along with psittacosis. The two diseases can be distinguished by the difference in clinical course and by the complement-fixation test for psittacosis. Supporting evidence for a final diagnosis of parakeet dander pneumonitis can be made by skin testing, presence of precipitin antibodies in an Ouchterlong gel diffusion test, positive hemagglutination test, and complete clearing of symptoms after removal of parakeets from the patient's envirnoment.

Parakeet dander pneumonitis is characterized by recurrent attacks of pneumonitis, acute bronchitis progressing to obstructive pulmonary disease, and pleural effusion. One affected patient had 400 parakeets in his basement, which continually exposed him to quantities of fine oily parakeet feather dust.—*Bull. Mason Clinic (Seattle), 14, (Dec., 1960): 127.*

Histopathologic Changes in Xanthomatosis in Chickens

J. H. Greve, D.V.M., M.S., and H. E. Moses, D.V.M., M.S.

IN 1955, cutaneous xanthomatosis was first described in chickens in New York[9]; occurrence was sporadic. There were gross skin thickenings, characteristic histopathologic changes, unimpaired egg production, and a failure to demonstrate an etiologic agent. In 1959, a similar condition was reported in chickens in Canada.

Since xanthomatosis is relatively new and uncommon and since it may be overlooked by poultry diagnosticians, these further observations on xanthomatosis in poultry from Michigan, Ohio, and Indiana in the period 1958 to 1960 are reported.

Materials and Methods

The chickens were submitted to the diagnostic laboratory at Michigan State University or at Purdue University when poultrymen noticed the unusual skin lesions. Suitable tissues were collected immediately after death and were placed in 10% formalin solution. Standard paraffin sections were cut at 6 μ and were stained with hematoxylin and eosin (H & E), hematoxylin and phloxin (H & P), or Mallory's trichrome. Frozen sections were cut at 20 μ and were examined unstained; then they were stained for lipids by flaming red dye or were stained for cholesterol after the modified Schultz method.[4] In frozen sections, crystals of both cholesterol and cholesterol esters were demonstrated by polarized light. Chemical analysis for total cholesterol content of affected and normal tissue was made.

Case Histories

Certain facts in the histories of all three cases were similar, but the significance of all items in the history has not been evaluated. All affected chickens were Single-Comb White Leghorn hens 7 to 24 months old. Five cases were concerned with one strain of Leghorns common to this area of the Middlewest. From this point, all flock histories seemed to deviate. The cases reported here involved birds on farms using litter, slatted floors, and range. The disease has been reported in caged hens.[10] The use of disinfectants and feed additives (arsenicals, fats, and antibiotics) also varied. In all cases, the number of chickens with xanthomas gradually increased in the flock. Egg production, egg quality, and general well-being of the flock were not affected. The degree of flock involvement with xanthomas ranged from 5 to 60%. In one instance, xanthomatosis appeared in successive flocks on the same premises. In other instances, the condition apparently did not recur after repopulation.

Gross Lesions

Xanthomatosis was recognized in these birds by gross cutaneous thickenings in the wattles, breast, abdomen, and thighs. The epidermis was an orangish yellow. On cross section, the tumor-like lesions were yellowish, firm, and adherent to the surrounding tissues. Amber-colored fluid drained from the cut surfaces. The feathers were broken or separated from one another, and they emerged from deeply dimpled follicles. These lesions were similar to those previously reported.[2,9]

Histopathologic Findings

Microscopically, the xanthomatous swellings differed from bird to bird, but they all had qualitative similarities. Although the primary changes seemed to be in the dermis, the hypodermis was also involved.

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An infiltration with vacuolated lipoid-laden macrophages ("foam cells") separated the normal cutaneous structures from each other. Lymphocytes, in both diffuse and nodular accumulations, were characteristic and often numerous. Often the lymphocytic accumulations formed perivascular cuffs. At the edges of some xanthomas, cuffing of capillaries by foam cells occurred (Fig. 1). Heterophils were not regularly found.

A great amount of collagenous connective tissue served for the stroma. Bundles of connective tissue fibers and delicate individual fibers were present throughout every portion of the lesion and served as a latticework for the inflammatory cells and cholesterol crystals. A fascial sheet that appeared abnormally yellow, grossly, contained a loose accumulation of foam cells, but the underlying muscle appeared normal.

The epidermis was slightly stretched but otherwise normal. Feather follicles appeared to penetrate deeply, owing to the outward growth of the xanthoma. Coagulation necrosis and other degenerative changes were often seen in the center of the xanthoma.

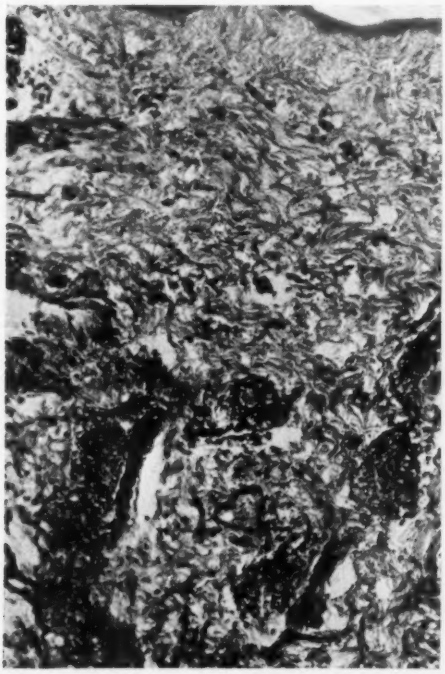

Fig. 1—The dark areas in the dermis represent dense accumulations (cuffings) of foam cells around small vessels at the edge of a large xanthoma. Mallory's trichrome stain; x 100.

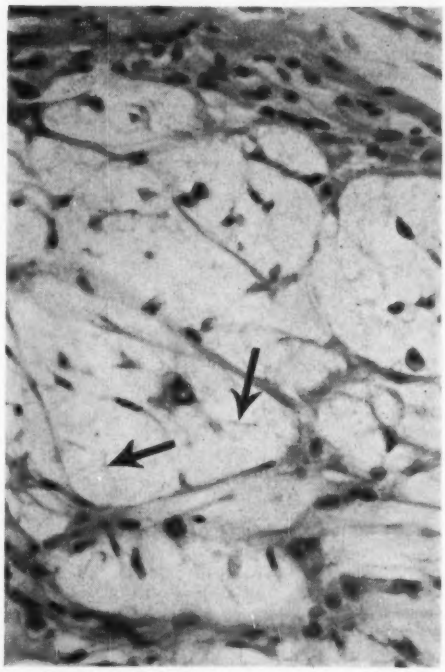

Fig. 2—Very small clefts grouped into tight clusters in the stroma of an xanthoma. Thin protoplasmic strands (arrows) are barely discernible between the tiny clefts. H & E stain; x 400.

Clefts representing cholesterol crystals were usually seen. In some birds, these clefts were small and were in tight clusters grouped into gross aggregates (Fig. 2). In others, the clefts were large and had a diffuse distribution, but often remained in loose clusters (Fig. 3). Usually crystals were surrounded by thin cytoplasmic films of multinucleated giant cells which apparently were attempting to esterize and resorb the crystals.[14] Cholesterol granulomas were thus formed.

Fluid-filled cysts were of 2 types: one type was lined by a simple squamous epithelium (Fig. 4); the other type had no epithelial lining and occurred in spaces between collagenous fibers. Some cysts were quite elongated, thereby resembling cystic lymphatic vessels. The cysts varied in size, from a few microns to a few millimeters in diameter. Generally, the smallest cysts

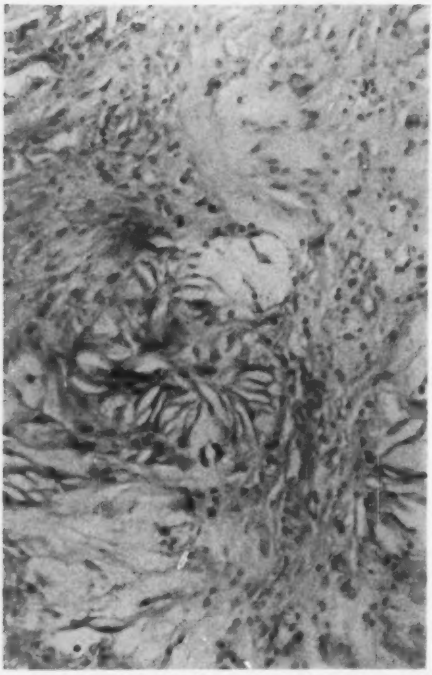

Fig. 3—Xanthoma made up of dense fibrous tissue, lymphocytes, reticuloendothelial cells, and large, loose clusters of cholesterol clefts. The borders of some clefts are outlined by the cytoplasm of macrophages. H & E stain; x 200.

were of the anepithelial type. The fluid within the cysts was eosinophilic and finely granular.

The relative quantities of the above changes were mixed together differently in each case. The histopathologic features varied markedly with each bird studied, *i.e.*, one had marked cystic changes, another primarily a foam cell infiltration (Fig. 5), and another little else but cholesterol crystals. The histopathologic variation was evident to some extent from area to area within a lesion as well as from bird to bird.

When flaming red dye was used, the frozen sections stained heavily for lipids. Analyzed by polarized light, numerous birefringent crystals (Fig. 6), characteristic of cholesterol, and some so-called "Maltese crosses" (Fig. 7), characteristic of cholesterol esters, were revealed. The cholesterol esters stained with the lipid stain, whereas the cholesterol crystals did not. A modified Schultz test[6] for cholesterol was positive in all cases. Small lipid deposits were found in the tunica media of the great thoracic vessels of 2 of 3 birds examined. Quantitative tests on xanthomatous tissue gave 1,270 mg. of cholesterol/100 Gm. of tissue.[7]

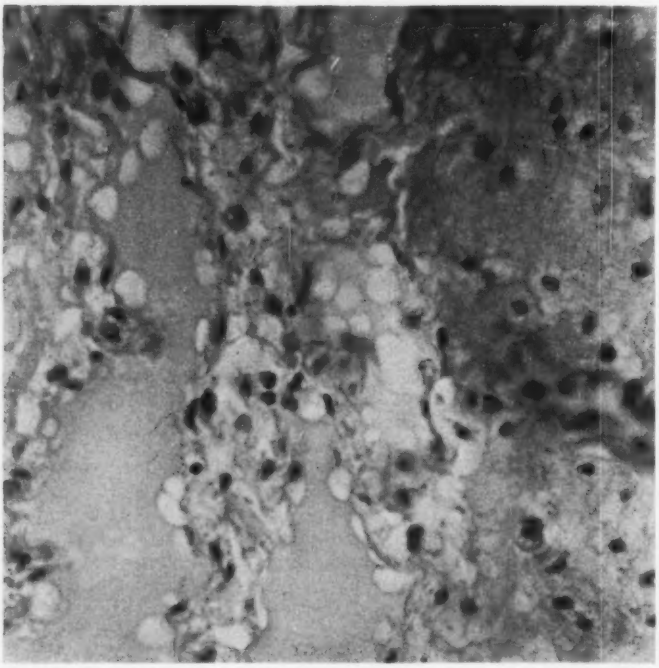

Fig. 4—A cyst lined with a simple squamous epithelium occurring in the stroma of an xanthoma. The cyst is filled with a finely granular, eosinophilic fluid. H & E stain; x 400.

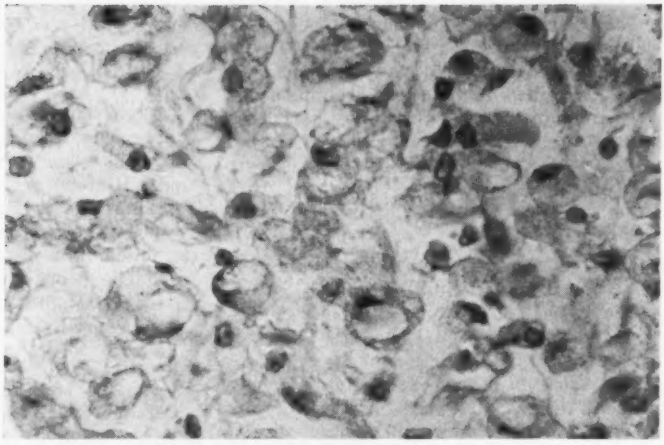

Fig. 5—A section of an xanthoma having primarily a foam-cell infiltration. Lipoid vacuoles of varying sizes are present within the macrophages. Scattered lymphocytes are also present. H & E stain; x 400.

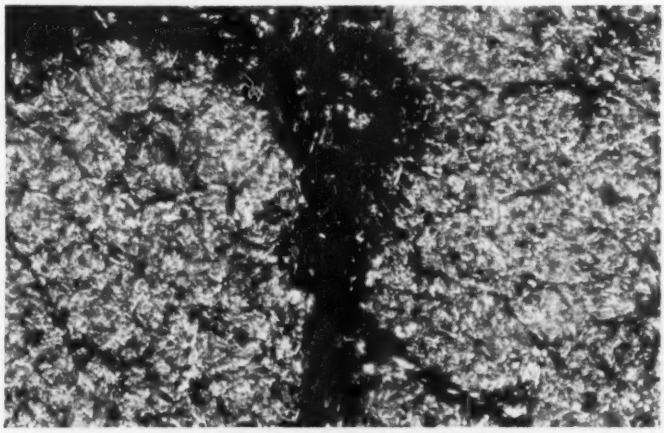

Fig. 6—The birefringency of the cholesterol crystals (white areas) and the inability of the stroma (black areas) to birefract is brought out by the analysis of polarized light. Unstained frozen section; x 100.

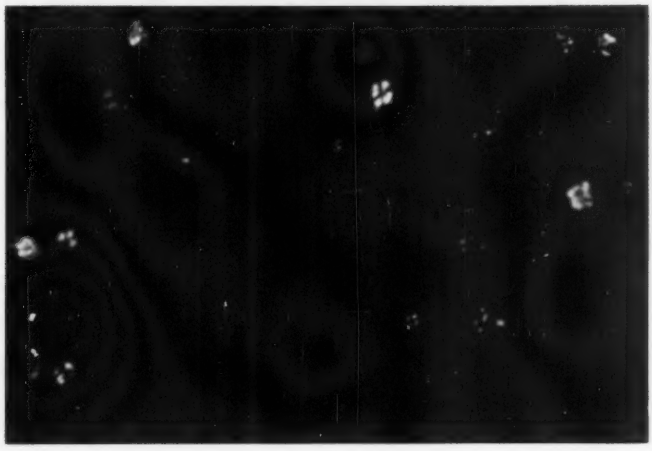

Fig. 7—So-called "Maltese crosses" visible during analysis of polarized light. These are characteristic of cholesterol esters. This is a field with more than the average number of Maltese crosses. No stain; x 400.

Normal values have been reported as being in the order of 150 to 200 mg. cholesterol/-100 Gm. of tissue.[2,9]

Although specific changes were not seen in other tissues, lymphocytic accumulations were commonly marked in many parenchymatous organs. Otherwise, ovaries and pituitary, thyroid, and adrenal glands were morphologically normal.

Discussion

Xanthomatosis is generally considered to be a disturbance of lipid metabolism of unknown cause leading to deposition of cholesterol and its esters in tissues. It is further characterized by lipid-laden macrophages (foam cells), other inflammatory cells, fibrous tissue, and multinucleated giant cells. Although the swelling may grossly appear neoplastic, the lesion is regarded as reactive.[10]

These observations are similar to those in earlier reports.[2,9] One investigator reported finding 4,282 mg. cholesterol/100 Gm. of tissue,[9] but the fibrous xanthoma tested by another[7] contained only 1,270 mg. cholesterol/100 Gm. of tissue. Apparently the total cholesterol content of xanthomas may vary.

Much of the experimental work on xanthomatosis has been done in rabbits. Trauma,[16] liver damage,[1] hormone imbalances,[3,15] diet,[4,5,11,12] and hypercholesterolemia[1] have all been suggested as causes or predisposing factors in the alteration of cholesterol metabolism. In poultry, hypercholesterolemia is not necessarily present in xanthomatotic chickens.[9]

The appearance of lesions of arteriosclerosis, another instance of lipid metabolic disturbance, is intensified by the presence of lymphomatosis in the bird.[8] Almost all xanthomatotic chickens studied here had marked lymphocytic accumulations in several organs. However, the interrelationships between these 3 conditions are not clear.

Our attempts to reproduce xanthomatosis in chickens have not been successful. Attempts by others[2,9] have also been unsuccessful. Malathion and pentachlorophenol were tested because of their use on some farms having xanthomatotic chickens. Contact with an affected bird, paintings of the skin with malathion or a pentachlorophenol-containing wood preservative, and exposures to fumes of this same wood preservative were all unsuccessful. Weekly topical application of the wood preservative caused an exudative dermatitis, but this regressed when applications were stopped.

Summary

Large cutaneous swellings occurred in the breasts, thighs, and wattles of White Leghorns under various management conditions. Histopathologically, xanthomas were characterized by lipoid-laden macrophages, lymphocytes, connective tissue, fluid-filled cysts, cholesterol crystals, and degeneration. The proportions of these alterations varied. Attempts to produce xanthomatosis were unsuccessful.

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Q Fever in Illinois—1958 to 1960

Prevalence of Reactors and Infections in Livestock

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Carl A. Brandly, D.V.M., M.S.

Q FEVER in man has been reported in Iowa,[7,18] Wisconsin,[3] Nebraska,[10] and Ontario.[11] An epidemic has been reported in Illinois.[12] Substantial serologic evidence now available indicates that *Coxiella burnetii* infections are numerous in livestock areas of the Midwest.[7,15]

The disease has been recognized as a public health problem in the United States since 1947, but its epidemiology in the Midwest is not clear. The usual syndrome in man is not sufficiently distinct for the physician to diagnose the disease without laboratory tests; morality is low.[1] Signs of disease in lower animals in the United States have not been reported. Whether the absence of the disease in man and the absence of reported cases in man in the presence of a known animal reservoir indicates failure to detect the disease, inapparent infections, or epidemiologic barriers, or all three, needs to be determined.

Since 1957, the College of Veterinary Medicine has been investigating a large reservoir of infection in certain species of livestock, primarily dairy cattle.

Materials and Methods

Serum samples of cattle and swine obtained for routine brucellosis and leptospirosis testing of herds in many Illinois counties were available in substantial numbers. Sheep make up only about 6% of the livestock of Illinois and are widely distributed in small flocks. Sheep serum samples were obtained from 17 counties representing the major geograpical regions of the state.

The serum samples were tested largely by a capillary-agglutination (CA) test,[5,6,9] although the complement-fixation test (CF) was used for all preliminary work in 1957 and later as a confirmatory test. The CA test was preferred because it has been found more sensitive[2] and more reliable than the CF test.[13,16] It has been stated "the serologic reactions obtained with Q fever antigens are highly specific and no cross-reactions occur with any of the other rickettsial agents nor with a variety of viral agents that have been tested."[4]

Milk samples from dairy cattle with serum positive to the CA test, were tested by the CA test, and attempts were made to isolate *Coxiella burnetii* by inoculation of milk into hamsters or guinea pigs. Composite milk samples from the 4 quarters of individual cows were collected in sterile tubes, 13 by 130 ml., packed in ice, and shipped directly to the laboratory. One or 2 ml. of raw milk was inoculated intraperitoneally into 350-Gm. guinea pigs or 50-Gm. hamsters. Blood samples were collected from guinea pigs and hamsters by cardiac puncture when received from the supplier, and their serum samples were tested for this *C. burnetii* antibody by the CA test; animals having a positive reaction were discarded. The guinea pigs were maintained in Horsfall-Bauer units. All air leaving the unit was directed through a Cambridge absolute filter.* Necropsy of the animals was done in a plastic hood connected to the absolute filter. The clothes worn in the unit were decontaminated in a garment sterilizer. The latter is essentially an asbestos board chamber measuring 6 by 3 by 2 ft. The temperature in the chamber was maintained at approximately 150 F. by heating elements controlled by a thermostat. Outer garments and respirator masks were stored in this chamber. Individual American Optical R2033 respirators of the type employed at Fort Detrick Biological Laboratories were worn by all personnel while in the laboratory room. The chief disinfectant was 4% lysol solution which was used in foot baths and for disinfecting surfaces.

During the initial trials, blood was collected and spleens removed from the animals 30 days after inoculation. The CA test was used to screen the serums for positive samples. The spleens were frozen at −8 C. or triturated in sterile broth and again inoculated into guinea pigs.

A new procedure was followed when isolation of *C. burnetii* proved difficult. Usually, 2 guinea pigs

From the College of Veterinary Medicine and the Agricultural Experiment Station, University of Illinois, Urbana. This research was supported by NIH grant E-2084.

*Cambridge Filter Corp., Syracuse, N.Y.

were inoculated with one milk sample; 1 guinea pig was killed 10 to 12 days later, and its spleen was removed for freezing or repassage. Blood was collected from the remaining guinea pig 30 days later, and the CA test was performed on its serum. Rectal temperatures were taken daily. To avoid possible transfer of infection via the thermometers, a separate thermometer was used for each animal. The thermometers were kept for 24 hours in lysol and rinsed in three changes of distilled water before use. Uninoculated control guinea pigs were placed in Horsfall-Bauer units with infected guinea pigs; some were also kept in open cages in the isolation room.

In cleaning the units, feces were heavily sprayed with fuel oil before removal to reduce the hazards of an aerosol. All waste matter was disposed of in heavy seed bags which would allow autoclaving. The outside surface of the bag and the receptacle used for transporting it were disinfected with a 4% lysol solution; the bag was either autoclaved or incinerated. Floors were swabbed with lysol and the interiors of the isolation room and Horsfall-Bauer units were sterilized with steam before new animals were introduced. Hamsters were kept in a separate building. Ordinary 5-gal. lard cans were utilized to hold them in isolation as previously described.[2]

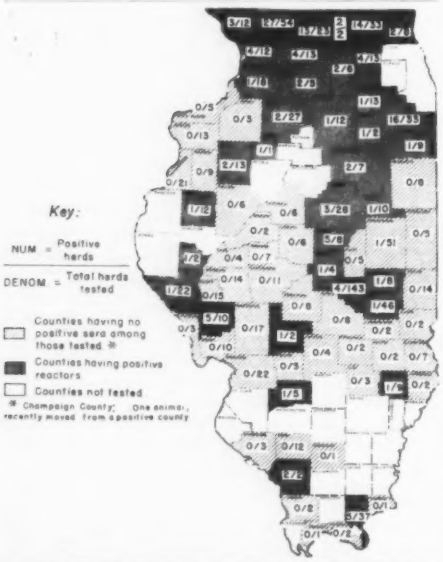
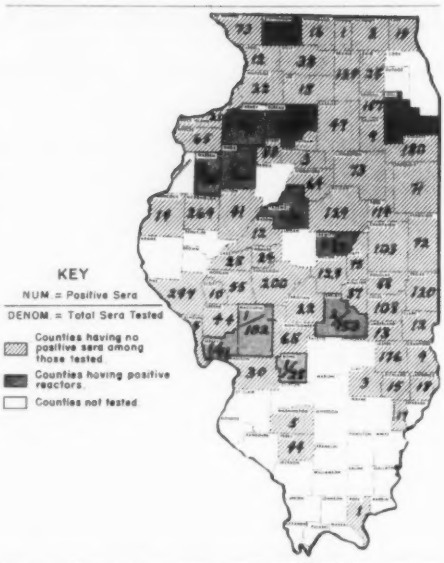

Key:
NUM = Positive herds
DENOM = Total herds tested
Counties having no positive sera among those tested. *
Counties having positive reactors.
Counties not tested.
* Champaign County: One animal, recently moved from a positive county.

Fig. 1—Reactors to *Coxiella burnetii* among Illinois dairy cattle as measured by the capillary-agglutination test (Luoto); state owned herds are not included. Total number of herds tested—887; total number of serum samples tested—8,575; total number of herds positive—141 (16%); total number of serum samples positive—594 (7%).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DENOM. = Total Sera Tested
Counties having no positive sera among those tested.
Counties having positive reactors.
Counties not tested.

Fig. 2—Reactors to *Coxiella burnetii* among Illinois swine as measured by the capillary-agglutination test (Luoto). Total number of herds tested—541; total number of serum samples tested—4,733; total number of herds positive—13 (2.4%); total number of serum samples positive—16 (0.3%).

As a further safety measure, all persons directly connected with the project were vaccinated with a purified, formalized, egg-adapted vaccine.* The California (AD) strain was used to prepare the vaccine, which was supplied as a 2%, infected, yolk-sac suspension, having 0.008 mg. of total nitrogen and 10 complement-fixing units (CFU) per milliliter. Two doses of the vaccine containing 1 CFU per dose were sufficient to protect guinea pigs against challenge with 10,000 minimal infectious doses of *C. burnetii*.

A skin test was used to determine whether the persons were sensitive to the vaccine. The skin test inoculum was prepared and the test performed by diluting the vaccine 1:50 in sterile saline solution and injecting 0.1 ml., intradermally. The test reaction was read at 48 hours and one week. A positive reaction was recorded when the erythematous area measured at least 2 mm. in diameter, with little edema. All who were negative to the skin test were given two inoculations of 1.0 ml. each, a week apart, followed by a "booster" dose (0.2 ml.) in six months. Persons were skin tested again before the booster dose was given. Serum samples from each person were tested for Q fever antibodies by

*Provided by Dr. Lauri Luoto and Dr. David Lackman of the Rocky Mountain Laboratory, Public Health Service, Hamilton, Mont.

the CA and CF tests (when possible) before vaccination and at one and six months postvaccination.**

Results

Results of the various surveys are shown graphically (Fig. 1-3). More than 8,500 dairy catle serum samples from nearly 900 herds in 78 of the 102 counties in Illinois were tested. Serum from 141 (16.0%) of the herds and 594 (7.0%) of the cattle was positive (Table 2). Serum was obtained from 78 counties. In 37 of these, there were positive herds; in 41, there was none.† The major Q fever region in Illinois consists of a solid block of 20 counties across the northern part of the state.

A total of 4,733 swine serum samples from 541 herds in 67 counties were tested. Only 13 (2.4%) herds and 16 (0.3%) serum samples in 12 counties were positive. In serum samples from 862 sheep obtained from 29 flocks in 18 counties, only 2 reactors were found. Both were native to Illinois.

Composite herd milk samples from four counties (Will, Kankakee, Grundy, and Kendall) were tested. Milk from more than 60% of over 300 herds was positive to the CA test. Isolations were made from 3 of 15 bulk milk samples tested by hamster and guinea pig inoculation as indicated by positive CA or CF tests on their serum samples 30 days after inoculation.

Spleen triturates of guinea pigs killed at 10 to 12 days (but not at 30 days) after inoculation resulted in development of CA titers in a second animal. Guinea pigs used in field isolations usually begin to shed the organisms in large numbers only after several guinea pig passages. Consequently, guinea pig passages were halted at two or three, and the spleens were frozen for testing when more secure isolation facilities would become available.

Results of tests on individual cattle of 4 herds from northern and central Illinois are shown (Table 1). Fifty-five (33%) of 167 serum samples were positive to the CA test; approximately 24% of the milk samples from these herds was also positive. Seven of 19 samples inoculated into laboratory animals produced positive CA titers at

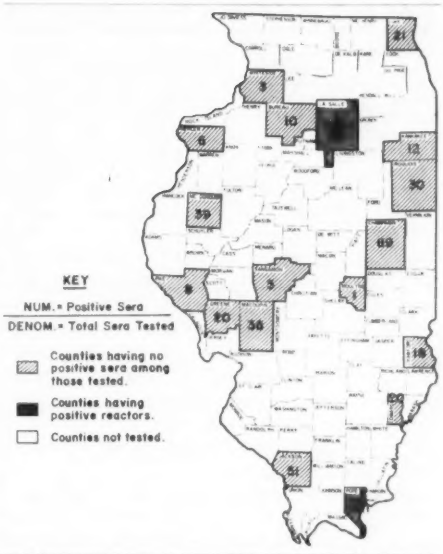
KEY
NUM. = Positive Sera
DENOM. = Total Sera Tested
Counties having no positive sera among those tested.
Counties having positive reactors.
Counties not tested.

Fig. 3—Reactors to *Coxiella burnetii* among Illinois sheep as measured by the capillary-agglutination test (Luoto). Total number of flocks tested—29; total number of serum samples tested—862; total number of flocks positive—2; total number of serum samples positive—2.

30 days. In the guinea pigs, there was no elevation of body temperature during this period. No lesions were observed at the site of inoculation.

In infected guinea pigs killed at 10 to 12 days and at 30 days, there were no pathologic changes. The spleens were not greatly enlarged, and other organs appeared normal in size and appearance. Rickettsiae were not seen in impression smears prepared from the cut surfaces of the spleen and stained by Machiavello's method. The results were similar to those obtained in Wisconsin.[3]

TABLE 1—Demonstration of Milk and Serum Antibody* for *Coxiella burnetii* and Isolation of the Organism from Cattle of 4 Dairy Herds**

		CA test results					
		Serum		Milk		Isolations	
Herd (No.)		+	−	+	−	+	−
A	65	26	39	11	26	1	3 (hamster)
B	37	7	22	5	32	1	6 (guinea pig)
C	35	7	28	4	25	2	2 (guinea pig)
D	30	15	15	12	18	3	1 (guinea pig)
Total	167	55	104	32	101	7	12

*As measured by the capillary-aggutination test.[5] **Criterion is a positive CA or CF test 30 days after inoculation of animals with 1 to 2 ml. of milk. Serial passage in guinea pigs only was accomplished by transfer of splenic material 10 to 12 days after inoculation.

**Instructions for therapeutic measures employed with human volunteers subjected to Q fever were supplied by Dr. Paul Kadul of the Army Biological Laboratories, Fort Detrick, Md.

†Since this survey was completed, positive herds have been found in both DuPage and Cook counties.

TABLE 2—Reactor Rates of Serum Samples of Illinois Dairy Cattle to *Coxiella burnetii* Antigen*

Period ending	Total No. serum samples**	Total positive	
		(No.)	(%)
12/1/58	3,313	160	6.5
1/9/59	5,886	305	5.7
2/10/59	7,758	506	6.5
4/6/59	8,575	594	6.9

*As measured by the capillary-agglutination (CA) test.
**Totals shown are cumulative.

One control guinea pig out of approximately 30, placed in the Horsfall-Bauer units during these trials, developed a CA reaction. Three of approximately 100 guinea pigs, obtained from commercial breeders, were positive when received and were eliminated. None of the control animals in open cages in the Q fever isolation unit or the animals in the separate holding buildings developed positive CA reactions.

One student assistant, who had been working in the Q fever unit while animals were being inoculated and observed, was hospitalized with a fever and severe headache in September, 1960. His CF titer was 3+ at 1:32 one month after vaccination. Blood samples taken at 30 days after hospitalization had the same CF titer. A chest radiograph taken during hospitalization was negative, and he had no cough or other evidence of respiratory involvement.

Discussion

The northern tiers of Illinois counties constitute the major reservoir of *C. burnetii* in dairy animals (Fig. 1). In general, there was a correlation between the number of dairy cattle in a county and the prevalence of reactors. In most of the northern counties, the ratio of positive cattle remained high during the entire survey; for instance, the ratios of positive-to-negative herds in Will County for the three periods beginning with the last quarter of 1958 were 9:15, 13:27, and 16:33 (Table 2).

This survey indicates that Illinois now has a much larger proportion of dairy cattle infected with *C. burnetii* than those reported from Wisconsin in 1956[3] and from Iowa in 1958.[16] Of 22,304 Wisconsin dairy cattle, 1.9% was positive for *C. burnetii* antibody; 22.3% was the highest percentage of reactors in a single county. Of 11,779 Iowa cattle, 0.71% had positive serum samples; 10.0% was the highest percentage of reactors in a single county.[16] Four northern Illinois counties and 1 west central county had reactor rates of 50.0% or above.

It has been suggested that Q fever may be spreading at an accelerating rate.[7] Therefore, it is not possible to say that Illinois has a higher Q fever infection rate among its dairy cattle than do two neighboring states since the rates in the latter may have increased during recent years. Evidence of a seasonal rise in reactors during the seven months of one study has been reported.[16] No such variation was found in our study (Table 2).

Q fever in man has not been adequately investigated in Illinois. About 23% of 37 veterinarians in 1958 and about 10% of 100 veterinarians in 1959 were positive to the CA and CF tests. The absence of reported cases in man in Illinois may be due to failure of physicians to diagnose the disease, inapparent infections, strains of low pathogenicity, or unknown reasons.

From the data, it is not clear whether the laboratory assistant mentioned previously had contracted Q fever. If he did, exposure might have occurred from cleaning contaminated Horsfall-Bauer units in spite of the precautions taken or from sources in eight dairies where he collected milk samples.

The survey casts some new light on a previously reported occurrence in Illinois.[12] Calves and sheep were both being processed in the abattoir involved. It is doubtful that sheep from the Midwest were a cause. The calves were young weanlings from 200-mile radius. It is probable that many were from *C. burnetii*-infected dams. Contaminated fluid and fetal memberanes could have contaminated body surfaces and alimentary tracts of the calves. *Coxiella burnetii* would have been viable for a period after parturition and present in large numbers on the hair and body surfaces and possibly in the alimentary tract and excreta of the calves.

Summary

The serum samples of 8,575 dairy cattle, 4,733 swine, and 862 sheep in Illinois were tested for *Coxiella burnetii* (Q fever) antibody, by the capillary-agglutination (CA) test. Serum from 16.0% of the dairy herds and 7.0% of the cattle was positive. Only 0.3% of the swine serum samples representing 2.4% of the herds reacted positively.

Serum samples from swine in 7 counties were positive although no reactors were found among the dairy cattle tested in those counties. Only 2 of the 862 sheep serum samples were positive; both sheep had been raised in Illinois. All three northern tiers of counties had reacting herds; the Q fever reactor rates were high as compared with previous findings in Wisconsin and Iowa. Except for a few counties near St. Louis, there were fewer reactors in the central and southern counties. The concentration of dairy animals in the St. Louis milkshed is higher than in other regions of central Illinois. Many bulk and individual milk samples from northern and central Illinois gave positive serologic reactions. Milk from these herds induced CA antibody responses when injected into hamsters and guinea pigs. The agent could be passed serially in guinea pigs by inoculation of infectious material at 10 to 12 days, but not at 30. Inoculation of milk into susceptible hamsters and guinea pigs caused positive CA and complement-fixation (CF) reactions in both species but no febrile response in guinea pigs.

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Poison Ivy Extract for Testing Antipruritics

A method for evaluating antipruritic drugs has been developed which makes use of the reaction produced by urushiol, the active principle of the poison ivy plant. A skin lesion is created in persons sensitive to poison ivy by application of a diluted solution of urushiol. Their response to antipruritic drugs can then be determined.

In a test with volunteers, methdilazine (Tacaryl—Mead Johnson) and trimeprazine (Temaril—Smith, Kline, and French) were compared. Both drugs had antipruritic efficacy but methdilazine produced the better response. Both are phenothiazine derivatives.—*Clin. Pharmacol. & Therap., 2, (Sept.-Oct., 1961): 605.*

Thallotoxicosis

John E. Willson, D.V.M.

THALLIUM is a toxic, heavy metal. It has an atomic weight similar to that of lead and of mercury, and it has many toxic and pharmacologic properties in common with these metals.

Poisoning in man has resulted from industrial exposure, application of thallium as a depilatory, and accidental ingestion of rodenticides or insecticides containing the metal. Thallium has also been used for suicidal and homicidal purposes.[11,24,32]

Fortunately, thallium has all but been abandoned as an internal medicament, and its use in cosmetic depilatories has largely disappeared. As a result, the incidence of thallium poisoning in persons has been markedly reduced. Today, thallium is used principally in the control of rodents and insects. The most commonly available salt of thallium is thallium sulfate, but thallium acetate has also been employed in pesticidal formulations. Other salts, such as carbonate, nitrate, and oxide, are used mainly in industrial chemistry.[23]

Thallium sulfate is used as a rodenticide or insecticide in the form of a syrup, jelly, or paste without any other ingredient; or it is applied to foods such as cookie or cake crumbs, peanut butter, or grain. The sweetness of these baits, coupled with the availability of the bait containers, certainly contributes to the hazards of thallium—especially for small children and household pets. Commercially available pesticidal preparations of thallium sulfate (ant traps, ant bait, and rat poison) usually contain 0.5% to 1.0% of thallium.

Thallium toxicosis, either experimental or accidental, has been reported in horses,[4] cattle,[4] sheep,[26] and cats.[28] There are numerous reports of thallium poisoning in dogs.[15,16,19,20] Thallium is toxic to plants and also prevents the germination of seedlings.[30]

In the ten years from 1949 to 1958, thallium poisoning represented approximately 8% of all suspected poisoning cases seen at the Angell Memorial Animal Hospital (592 poisoning cases, 50 cases of thallium poisoning). Of these 50 animals, 49 were dogs, 28 of which died, and 1 was a cat which died.[12]

Absorption, Distribution, and Excretion

Thallium compounds are readily absorbed from the gastrointestinal tract and through the skin. Peak blood levels in man are reached two hours after oral administration.[31] Thallium salts are fairly generally distributed throughout an animal's body. At necropsy, the highest concentrations of thallium per unit weight are found in the kidneys, pancreas, spleen, and muscles.[7] Thallium is found in all body fluids. It is excreted by both the kidneys and the intestine in approximately equal amounts.[18] It passes into the fetus; the milk of poisoned animals may be toxic to the suckling.[30]

Thallium is retained in the organs for long periods. It has been estimated that approximately 45% of an administered dose was still present in the human body 24 hours after administration.[31] Like lead, thallium is excreted slowly, and it has been demonstrated in the urine months after administration. In dogs, 62% of the amount given orally was excreted in the urine in 36 days; in rabbits, 70% was excreted in the urine and 14% in the feces within 31 days.[30]

Toxicity

The fatal dose in man ranges from 0.2 to 1.0 Gm.[3,32] Other reports place the fatal dose for adults at between 5 and 6 gr., or

*Dr. Willson is a member of the staff of the Angell Memorial Animal Hospital, Boston, Mass.

approximately 6 to 8 mg. per kilogram.[27] The minimum lethal dose in dogs is from 12 to 15 mg. per kilogram of body weight.[7] As for the toxic salts (thallium acetate), the minimum lethal dose for man and animals lies between 15 and 25 mg. per kilogram of body weight.[4,13] For some unknown reason, toxicosis in man is believed to be distinctly lower before puberty.[30]

Clinical Findings

The signs of thallium poisoning, both in animals and in man, are somewhat similar and are determined largely by the amount of thallium absorbed into the body.

Thallium acts as a general cell poison with cumulative properties and a variety of toxic manifestations. In dogs, three types of clinical cases can be distinguished—peracute, acute, and chronic.

The peracute form is seen when large amounts of thallium are ingested. It is characterized chiefly by hemorrhagic gastroenteritis, colic, vomiting, and trembling. These are followed by motor paralysis, dyspnea, and death in 24 to 40 hours.

The course of the acute form is slower, lasting several days to a few weeks. The prominent signs are gastrointestinal colic, vomiting, diarrhea, stomatitis, conjunctivitis, excessive salivation, pain in the extremities, muscular tremors, convulsions, paralyses, bronchitis, and bronchopneumonia. Ecchymotic and petechial hemorrhages are also observed. In fatal cases, death can occur in a few days, or be delayed several weeks, with severe nervous signs and respiratory failure developing terminally. Blindness and other paralyses of the special senses may develop. If the dose was not too large or too long continued, the recovery may be complete, but this may require many months. Permanent sequelae such as kidney disease and ataxia are not uncommon.

The signs of chronic poisoning are similar to those of acute poisoning but are generally milder. Endocrine disorders may occur in addition to the digestive and nervous disturbances.[30]

All of the aforementioned signs are not characteristic of thallium poisoning alone, but conform generally to those of any heavy metal poisoning. The most common and characteristic signs of thallium intoxication are hair and skin changes. These signs take one to three weeks to develop and are absent in peracute and many acute cases. Apparently, they are not pathognomonic.[32] Diffuse alopecia or actual necrosis of the superficial layers of the skin may appear. The necrosis is often preceded by reddening of the skin, especially in areas subject to friction. Thickening of the skin and hyperkeratosis around the mouth, eyes and foot pads also occur. The web of skin between the foot pads swells and oozes serum.[21] Usually, alopecia develops in many areas of the body, particularly the ears and friction surfaces, and the hair can be pulled out with relative ease. In the more acute cases, the visible mucous membranes are often brick-red to mahogany in color.[20]

The mechanism of hair loss is not known,[32] but it may be due to the effect of the metal on the autonomic nervous system.[9] This action of thallium is reversible and, unless the dose is too large, the normal growth of hair will be resumed.

Depilation is apparently not a prominent sign in cats.[28] In horses, sheep, and cattle the main signs are excessive salivation, dysphagia, and depression. In sheep, shedding of the wool occurs.[4]

Usually, there are albuminuria and an increase of cells and casts in the urine sediment.[3,8] Blood studies indicate leukocytosis with neutrophilia.

Necropsy Findings

Thallium induces degenerative changes in all cells. The necropsy findings vary with length of illness and severity of intoxication. In acute poisoning, there may be no characteristic lesions.[2,7]

Usually there are severe gastroenteritis and inflamation of the respiratory and lower intestinal mucosae. Keratitis may be present. The gastroenteritis tends to become ulcerative. Subserosal hemorrhages may be found. The kidneys are usually severely congested.[29]

Severe degeneration, with necrosis of the liver parenchyma and myocardium, has been described. Petechial hemorrhages, perivascular cuffing, and demyelination occur in the brain.[15]

Histologically, in the depilation stage, the hairs appear split and broken, and the hair follicles are often enlarged. There are also changes in the keratinization of of the epithelium and scaling.[30]

Diagnosis

The clinical diagnosis is based on the history, clinical signs, possible access to thallium, and certain confirmatory tests.

A specific test for the identification of thallium has been described.[33] One drop of urine is placed on a test plate. To this is added 1 drop of 0.4% nitrate of bismuth in 20.0% nitric acid, followed by 1 drop of 10.0% sodium iodide. A red precipitate develops if there is a significant amount of thallium in the urine. One drop of an aqueous saturated solution of sodium thiosulfate will increase the specificity of the test if certain other ions are present in the urine (Pt^{++++}, Fe^{+++}, and Ce^{++++}).

A urine test involving paper chromatography has been described.[22] Other more complicated chemical and spectrographic analytical methods are available for testing body fluids or excreta.

Methods exist for the detection and the quantitative determination of thallium in biologic material.[5,6] In fatal acute poisoning in man, the thallium content may range from 0.5 mg. to 10.0 mg. in 100.0 Gm. of tissue.[7]

Treatment

General emergency measures such as emesis, gastric lavage, and catharsis may be helpful if used within an hour or so after ingestion.

Unfortunately, there is no known specific antidote for thallium, but a large number of treatments have been recommended. Sodium iodide solution (0.3 to 1.0 Gm.) injected intravenously in an attempt to immobilize the thallium has been suggested as a treatment for thallium poisoning in dogs.[25] However, it is reported that no evidence exists that iodides materially affect the course of poisoning once significant amounts of thallium have been absorbed.[32] Oral or intravenous injections of sodium thiosulfate solution in doses of 0.3 Gm. daily have been used in an attempt to eliminate the thallium.[25] The efficacy of this treatment has also been questioned.[9,32] Dimercaprol (BAL) was reported to be an effective antidote in dogs, if treatment was started before the appearance of pronounced clinical signs. However, it was ineffective in cats,[17] and it did not enhance the excretion of thallium in controlled experiments with rats.[18]

Diphenylthiocarbazone* is an effective antidote against thallium in rats.[18] When given orally, it forms a firm complex with thallium and aids in the elimination of the metal through the urinary tract and the intestines. In addition, it apparently penetrates into the cells and is able to detoxify the thallium deposited in the organs. The drug has produced diabetes mellitus in rabbits,[14] but it was not an effective diabetogenic agent in dogs.[1] It also has a goitrogenic effect on rats.[13] For these reasons, it should be used with caution. Diphenylthiocarbazone has been used in treating canine thallotoxicosis in doses of 70 mg. per kilogram of body weight three times a day.[20]

Experimentally, activated charcoal and potassium chloride, both given orally in aqueous suspension, increased the excretion of thallium in the feces (activated charcoal) and in the urine (potassium chloride).[18] In addition, both of these substances lowered the toxicity of thallium and are easily available and harmless. Potassium chloride (2 to 6 Gm. daily) has been used in dogs.[20]

Supportive measures such as sedatives, fluid therapy, and vitamins are useful. Soft, protective bedding and adequate nutrition are essential. The administration of sulfur-containing amino acids, such as cystine and methionine, decreased the mortality rate in acute poisoning and increased the survival period in chronic thallium poisoning in rats.[10]

Summary

Thallium poisoning continues to be reported, despite the decreasing use of this highly toxic metal. Thallium is readily absorbed from the gastrointestinal tract and is retained in the body for long periods. Alopecia and skin changes are the most characteristic signs of thallium toxicosis.

Thallium is excreted by both the kidney and the intestine. A specific test for the identification of thallium in urine consists of adding 1 drop of 0.4% nitrate of bismuth in 20.0% nitric acid to 1 drop of urine on a test plate. One drop of 10.0% sodium iodide is added to this mixture. A red precipitate develops in the presence of thallium.

*Dithizone, Distillation Products Industries, Eastman Kodak Co., Rochester N.Y.

There is no known specific antidote for thallium. Sodium iodide and sodium thiosulfate have been recommended as treatment for thallium poisoning, but their efficacy has been questioned. Diphenylthiocarbazone aids in the elimination and detoxication of thallium.

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Editorial

New Automobile Emblem

Occasionally a veterinarian wistfully looks at the automobiles and commercial vehicles in his community and wishes he, too, could make his presence known by means of tasteful artwork and lettering on his automobile or ambulance. It's not surprising, then, that the AVMA committee on ethics and (since 1958) the AVMA Judicial Council have been asked to express opinion with respect to the propriety of signs on vehicles.

Vehicular Signs Taboo

In 1955, an ethics committee report stated "It is considered unethical for a veterinarian to have his name and title or that of his hospital appearing in letters on the side or on any portion of any vehicle appearing before the public." And a 1956 committee deemed it objectionable ". . . for any practitioner to use a motor vehicle upon the sides of which display signs of any character have been placed, such as a pet ambulance. This constitutes direct advertising and invites criticism by fellow practitioners."

The New Auto Emblem

The evils of direct advertising by professional people is one thing, but who will deny that a message carried on an automobile moving regularly about a community will reach many people? The solution is to present the message in a tasteful, ethical way.

For many years, the AVMA offered its members, at cost, an auto emblem designed to be fastened to the license plate bracket. With the progression of styling changes like tail fins and recessed license wells, however, it became difficult or impossible to mount the emblems.

Now, however, AVMA members may obtain an automobile emblem that has an adhesive backing. It is easily applied to the surface of the automobile and can even be bent somewhat to conform to body contours. Three inches in diameter, it is a buffed bronze metal with maroon and gold baked-enamel finish. The veterinary caduceus appears in the center in bronze, surrounded by the words "American Veterinary Medical Association." This emblem can be purchased for $2.50 from AVMA Membership Services.

Auto Emblems, Pro and Con

Why have some professional people shied away from auto emblems? Many physicians seem reluctant to label their cars in a way that might cause them to be pressed into giving medical aid under adverse circumstances at the scene of a highway mishap. That this reticence has no basis in documented legal proceedings is not germane to the present discussion. Suffice it to say that the veterinarian's position is different.

Some physicians feel that it isn't especially good public relations to clearly identify themselves with high-priced cars. Others feel that the medical emblem on an automobile invites thievery by narcotic addicts.

The disadvantages apply far less to veterinarians, however, and those who have displayed the professional emblem on their vehicles for years offer many reasons why they will continue to do so. The least important, in our estimation, is the consideration extended by law enforcement officials to the veterinarian who exceeds the speed limit in making emergency calls and finds it necessary to violate parking regulations when he gets there.

The PR Value

It's really the public relations value of the automobile emblem that prompts us in good conscience to urge veterinarians to use it. Here is an opportunity to present, in a perfectly ethical fashion, a tastefully restrained message to the public that the owner of the car is a professional man who belongs to his national association; and that he is a member of the medical community going about his job of contributing in some way to the public health and welfare.

We are a profession relatively small in numbers, but if all of us, practitioners or not, were to display the caduceus on our cars, many thousands of people every day would be reminded that veterinarians exist. People who have never had occasion to talk with a veterinarian might be encouraged to find out what he does, and some young people may be stimulated to inquire about the possibilities of a career.

The use of this attractive emblem has definite public relations value. Only a little effort is required to put it to work.

University Tuitions Rising

A survey of 97 colleges and universities from coast to coast indicated that the present academic year will cost 5.2% more than last year for tuition, fees, room, and board. According to the survey, the average per student cost for the 1961-1962 academic year will be $1,869 for tuition, fees, room, and board; the average cost the preceding year was $1,777. One registrar estimated that tuition in his university would be about $2,000 per year by 1970 compared with $1,155 this year.

A college graduate is still likely to earn more during his lifetime. A man with four or more years of college, based on 1958 economic conditions, has an estimated lifetime income-producing ability of $435,242. If he has only four years of high school, his lifetime average would be $257,557. It would be $181,695 for a man with an eighth grade education.—*AMA News (Oct. 2, 1961): 14.*

New Books

Animal Vision

This book is written for the general reader rather than as a detailed textbook on the subject of vision. It is informative yet written in a style that makes for enjoyable reading. The author has included information on many species of animals; therefore, it should be of interest to the veterinarian, the zoologist, or any one interested in biology. The author explains the actions of animals as reactions to objects observed in their environment. He also explains how the eye varies in position and structure in the various species, and how it is utilized in animals with nocturnal habits in contrast to those that move about under diurnal conditions.

The author does repeat some of the information in the different sections of the book but this is not a serious fault. The description of the ramptype retina in the horse and the manner in which the animal uses its eyes as it moves should be of interest to large animal practitioners. The descriptions of the eyes of zoo animals and some birds is not generally offered in a veterinary curriculum.

This book is for general information and is not a treatise on the eye. There is no attempt made by the author to discuss eye diseases or to advocate any type of therapy.

The author is well qualified in the field of veterinary ophthalmology, and the book should be a welcome addition to the library of anyone interested in biological sciences.—[*Animal Vision. By R. H. Smythe, M.R.C.V.S. 250 pages; illustrated with line drawings. Charles C Thomas, Springfield, Ill. Published simultaneously in the British Commonwealth of Nations by Herbert Jenkins Ltd., London, Eng. 1961. No price given.*]—CHARLES DIESEM.

Pharmacology of Anesthetic Drugs

This fourth edition of a recognized text supplies the reader with up-to-date information on the practical use of anesthetics. Anesthesia, like other branches of science and medicine, has advanced considerably since World War II. This book stresses the fundamental principles in such a way as to enable the practitioner to know not only what anesthetic to use, and where and how, but also why.

By use of labeled diagrams and well-organized tables, the author presents, in a concise manner, the important material relating to routes and methods of administration, routes of elimination, effects on organs, on the central nervous system, on respiration, on circulation, comparisons of drugs, uses, contraindications, advantages, and disadvantages.

The title belies the thoroughness with which the subject is treated for, in addition to the general anesthetics (both volatile and nonvolatile), the author discusses hypnotics, sedatives, analgesics, and local anesthetics. Likewise, many agents needed in conjunction with anesthetics, such as analeptics, muscle relaxants, tranquilizers, atropine, sympathomimetics, oxygen, and carbon dioxide, are discussed.

It should be more and more apparent that there is no "one best" anesthetic, or tranquilizer, or sedative. Although the book was written with the physician primarily in mind, all who use anesthesia, including veterinarians, will find here the information needed to make the most appropriate choice of drugs for a specific situation. For a complete compendium on anesthesiology, this 232-page volume is highly recommended.—[*The Pharmacology of Anesthetic Drugs. By John Adriani. 4th ed. 232 pages; illustrated Charles C Thomas, Springfield, Ill. 1960. Price $11.00*]—CLYDE F. CAIRY.

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WHAT IS YOUR Diagnosis?

Make your diagnosis from the pictures below — then turn the page ▶

Fig. 1—Radiograph, dorsovolar view, of the left forefoot of a Standardbred mare.

Fig. 2—Radiograph, lateral view, of the left forefoot of a Standardbred mare.

History.—A sound, 3-year-old, Standardbred mare was trotting slowly when suddenly she became frightened, lunged, and immediately became lame in the left foreleg. The lameness persisted, and the sole of the foot seemed sore. The horse walked soundly when the posterior digital nerves were blocked. Lateral and dorsovolar radiographs of the foot were taken (Fig. 1 and 2).

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Fracture of the navicular bone of the left forefoot.

Comment.—The fracture (Fig. 3 and 4) may have occurred because the bone was weakened as a result of bone resorption or aseptic necrosis from dis-

Fig. 3—Radiograph, dorsovolar view, of the left forefoot. Notice fracture of the navicular bone (arrows).

Fig. 4—Radiograph, lateral view, of the left forefoot. The navicular bone is fractured (arrows).

turbed circulation. Severe contusion resulting from impact with a hard surface may also be sufficient to cause such injury.

In most cases of navicular fracture, treatment is likely to be ineffective. Pain may be relieved by special shoes that will reduce pressure to the area.

This report was submitted by W. H. Rhodes, V.M.D., School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa., and was prepared with the assistance of Wayne H. Riser, D.V.M., M.S., Kensington, Md.

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CLINICO-PATHOLOGIC CONFERENCE

From the School of Veterinary Medicine
University of Pennsylvania

Presentation of the Case

*Dr. Olive Britt.**—A yearling Thoroughbred colt was foaled in England and, as a weanling, accompanied his dam to Italy where an illness was diagnosed as kidney disease. The colt and mare subsequently were returned to England. No further information was available until the animal arrived at the present owner's farm in the United States on Sept. 9, 1960. On September 11, the colt appeared to be ill. Improvement was noticed the next day, and the animal seemed normal until September 18 when it again became ill and did not improve until Septemer 25. On September 26 and 27 it ate well but, during the night of the 27th, the colt became uncomfortable again and showed signs of intermittent colic and bloat.

The colt was admitted to the University of Pennsylvania Large Animal Clinic on Septmber 29. Its rectal temperature was 100.2 F., pulse 42, and respiratory rate 20. There was slightly increased intestinal peristalsis. A rectal examination at this time was attempted but, due to a large amount of gas, was not completed successfully. The general appearance of the colt was poor.

Prepared by:

R. S. Brodey, D.V.M., M.Sc. (V.S.)

Assistant professor of Veterinary Surgery

J. E. Prier, D.V.M., Ph.D.

Associate Professor of Virology

LABORATORY DATA

	September 29	October 1
Red blood cells	8,750,000	7,320,000
White blood cells	6,900	10,400
Lymphocytes	80%	60%
Neutrophils	17%	30%
Nonsegmented neutrophils	1%	----
Monocytes	2%	2%
Eosinophils	----	2%
Packed cell volume	35%	33%
Hemoglobin	12.9 Gm./100 ml.	11.2 Gm./100 ml.
Blood urea nitrogen (BUN)	35.5 mg./100/ml.	----
Icterus index	----	10
Sedimentation rate	----	10 min.-11 mm. 20 min.-37 mm.
BUN	----	46.7 mg./100 ml.
Sugar	----	108.0 mg./100 ml.
Total protein	----	2.9 Gm./100 ml.
Albumin	----	2.4 Gm./100 ml.
Globulin	----	0.5 Gm./100 ml.
A/G ratio	----	4.8:1
Fecal examination	No ova	No ova

On September 20, the animal appeared very depressed. His temperature was 102.1 F., pulse 40, and respiratory rate normal. Peristalsis was increased, particularly in the area of the cecum. The feces were not formed and a "fermentative" odor was apparent. A rectal examination was again attempted but, due to the animal's straining, was not successful.

The serum uric acid level was 6.9 mg./100 ml. The total serum protein was 2.8 Gm./100 ml., and the colt was given 1,000 cc. of 5% glucose in saline solution and 1,000 cc. of a protein hydrolysate solution** intravenously.

* Dr. Britt is instructor in veterinary surgery.

**Aminosol, produced by Abbott Laboratories, North Chicago, Ill.

On the day of admission, the urine was light amber and turbid, its specific gravity was 1.034, pH 6.5, sugar 4 plus, bile 1 plus, and it contained amorphous crystals. *Proteus sp.* sensitive to neomycin and methenamine was isolated from the urine. A urine sample taken, however, was negative.

On October 2, the colt appeared to be unchanged.

On October 3, he was still very depressed. For the first time, the mucous membranes appeared to be congested. Examination for blood parasites was negative. In the afternoon of the same day, the colt seemed to be in more distress, and an anticholinergic drug* was administered parenterally. A rectal examination was made after administering epidurally 5 cc. of 3% procaine. A firm nodular mass in the area of the small intestine and medial to the cecum was palpated. The full thickness of the rectal wall ruptured at this time. The colt was given 1.25 Gm. of oxytetracycline intravenously and this was repeated in 12 hours. Two quarts of mineral oil was administered by stomach tube. The serum uric acid was 8.85 mg./100 ml. The total protein was 3.1 Gm./100 ml., of which albumin was 2.82 Gm. and globulin 0.29 Gm. (A/G ratio 10:1).

On October 4, the animal was down and in severe distress. An anticholinergic drug* was administered parenterally. The body temperature was 101.6 F., pulse 66, respiratory rate 60, and peristalsis was very depressed. An exploratory laparotomy, using a low right flank approach, was performed after which the animal was euthanatized.

Clinical Discussion

*Dr. M. Mackay-Smith.***— When I am faced with a case which isn't too obvious, I try to break my differential problem down into two principal categories. The first is a systematic review of the history and symptomatology so that I know what system is involved principally. Second, I consider it on the basis of possible etiology. I think this case lends itself to this sort of analysis.

In this colt there is evidence of generalized disease, particularly if one considers the hypoproteinemia, fever, relative lymphocytosis on repeated hemograms, and elevated BUN and uric acid. There are other ancillary but less striking features. As far as the systems themselves are concerned, we have little evidence of respiratory or nervous system involvement. The urogenital findings, with the exception of the plus 4 sugar in the urine, can be disregarded. Although the animal was emaciated, this is probably evidence of generalized disease rather than of any particular musculoskeletal involvement. I think the evidence points most strongly to gastrointestinal disease and I'd like to come back to this because it seems to be the area where our attention should be concentrated.

As far as cardiovascular disease is concerned, there is no particularly overt sign, but any horse with chronic intermittent abdominal distress or colic may have a vascular problem. Last, I would consider the reticuloendothelial system. The near to almost absent globulin in this horse suggests to me that he is having problems in conversion and production of protein which may be directly associated with the tissues responsible for that function or may be associated with an intestinal problem.

Going back to gastrointestinal disease, I am inclined to divide them into two categories, the first involving primarily the intestines, and the second some abdominal disease outside the intestines. The character of the feces, the duration of the condition, and so forth, suggest that the disease in this colt might be in either category. There are two sources of sounds within the abdomen; either there is hyperproduction of gas, or else there is increased bowel motility. In this colt, there is some evidence that a good deal of gas is being produced; although he was apparently not having much trouble passing gas, a rectal examination was precluded on one occasion owing to the extreme amount of gas.

To summarize my review, I think that we have a general picture of metabolic starvation, exhaustion, and marked catabolism which is progressing at a rate which I would characterize as being subacute. The lapse of time between the apparent onset of the disease and our examination of the horse does not suggest a rampant process.

*Methamyprone, produced by Myers-Carter Laboratories, Inc., Phoenix, Ariz.

**Dr. Mackay-Smith is instructor in veterinary surgery.

My second consideration is that the hypoproteinemia resulted from decreased absorption from the gut, rather than any specific organic lesion in the liver. If the low protein levels were due to primary hepatic failure, I would expect to see other changes referable to hepatic disease such as jaundice, urinary changes, and so forth. Probably a disease process outside the intestine is having some effect on absorption.

I would continue my analysis according of the second category (etiology) in which I divide the factors into hereditary, metabolic, organic, traumatic, infectious, and neoplastic. There is no suggestion that this is an hereditary disease; the history isn't very good, but if the colt had been this sick since he was born, we probably would have such information. As far as primary metabolic disturbance, that is to say failure of intermediary metabolism with respect to enzyme systems and that sort of thing, I don't think it can be completely ruled out. I don't feel qualified to comment in any great detail on that. There are certain symptomatic features which reflect metabolic disturbances, but I think they are largely just symptomatic. Chances are that there is some mechanical or morphologic tissue change in the abdominal cavity. Before surgery, a mass of some kind was discovered in the abdomen, and it should be associated with the problem until proved otherwise. Trauma doesn't seem to play a particularly important role here; certainly there is no history of extrinsic trauma. I think that the mass in the abdomen suggests infection or neoplasm. The lymphocytosis suggests that there has been some chronic inflammatory problem, but it does not give any distinct information as to whether it might be a bacterial or parasitic problem or whether it might be secondary to neoplasia.

After reviewing the case from both these standpoints, we have a paraenteric mass which might be infectious or neoplastic, associated with generalized manifestations, and existing over a fairly long period. I would think that neoplasm would be the less likely of the possibilities. The thing that most often produces decreased absorption from the gut is a thrombus of the anterior mesenteric artery, and I think this is probably the direction in which one should lean as a presurgical impression. In this particular case, there seems to be no good means of making a more specific diagnosis without a laparotomy.

Dr. R. S. Brodey.—Does anyone else have any ~~comment~~ on the diagnosis?

*Dr. R. R. Marshak.**—In your differential diagnosis, Dr. Mackay-Smith, you didn't mention or try to explain the high blood urea nitrogen (BUN) and the uric acid.

Dr. Mackay-Smith.—I would be inclined to explain the high BUN on the assumption that this animal is probably using protein as a source of carbohydrate, the proteins coming from tissue catabolism.

*Dr. J. Deubler.***—The BUN was not determined at the time the uric acid was last determined (the day before the laparotomy). The condition of the animal was certainly much worse at that time, and the BUN could have been elevated by then. This might have had something to do with the uric acid elevation.

Dr. Mackay-Smith.—Well, when the BUN was first determined, it was a little higher than we generally consider normal. The next time it was up to 46, and at the last sampling, when the uric acid was determined, I would ascribe it to tissue catabolism and protein breakdown. The BUN seems more significant than the uric acid.

Dr. Marshak.—What is your thinking about the A/G ratio? You expressed yourself in terms of the low total protein, but why is the globulin in the A/G ratio so low?

Dr. Mackay-Smith.—I would be inclined to think that there was decreased production of globulin which we have been taught to associate with the reticuloendothelial system. I don't know why the ratio was so low, unless it's just from complete exhaustion of this metabolic mechanism.

Dr. Marshak.—You're drawing attention to one interesting thing about this case; that is, the laboratory data confuses the diagnosis tremendously, if you want to pay any attention to it.

Dr. Mackay-Smith.—Well, we all were confused, but Dr. Raker's presurgical diagnosis was made separately and all I can do is fall back on what I already know.

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* Dr. Marshak is professor of veterinary medicine.

**Dr. Deubler is assistant professor of veterinary pathology.

Dr. Marshak.—Well, my main point in bringing this up is to call the attention of the group to the variant laboratory data which I thought were possibly glossed over a little bit and were really very confusing.

Dr. Mackay-Smith.—I didn't mean to gloss over them, but I didn't feel that I should speak at great length about them. I'm sorry that I did have so little to say about them. I was hoping to hear, myself, some basis on which they could be correlated with what was found. When some of the lesions are discussed we might have more information.

*Dr. R. M. Sauer.**—I'm not sure what the normal BUN is in the horse.

*Dr. Raker.***—Ten to twenty.

Dr. Sauer.—Then it is probably high in this case, even if you do put it on the basis of protein catabolism. Now according to some, the A/G ratio normally is about 1 to 1, about 3.25 albumin and 3.25 globulin. My understanding is that in malnutrition due either to hypermotility of the bowel or possibly lack of absorption, if the protein level falls, the decrease is primarily in albumin. The globulin as a rule stays normal and in certain cases of infection may run high. Now the second record shows 0.28 for the globulin; is that right? Well, considering it to be 1 to 1 in the horse normally, this seems almost like an agammaglobulinemia and, on a dietary basis, I just cannot explain it one way or the other. If there were agammaglobulinemia on an hereditary basis, I think the animal should have been dead from infection long before this; so it doesn't add anything other than to drag a red herring across the trail. I can't explain this protein situation, or even believe it, frankly, on the basis of the figures that were shown.

Dr. Marshak.—I'd just like to make one other comment. One thing I thought about, especially in view of the abdominal mass, was the possibility of lymphoma, which would explain something that was left out of this history: the fact that on the subsequent fresh blood smears there were some abnormal lymphocytes. The high uric acid could go along with lymphoma, as the lymphocytes are mainly nuclei. As the nucleic acids are broken down in such tumors, high serum uric acid develops. In fact, cows with lymphosarcoma and other lymphomas have hyperuricemia.

*Dr. Sauer is assistant professor of veterinary pathology.
**Dr. Raker is professor of veterinary surgery.

Clinical Diagnosis

1) Parenteric lesion, probably thrombus of the anterior mesenteric artery.

2) Possible neoplasm.

Pathologic Findings

*Dr. Harry Fegley.**—On primary incision, we found a large amount of fluid mixed with fecal material (from the tear in the rectal wall) as soon as we opened the abdomen. The posterior 3 ft. of the colon and the anterior 3 ft. of the rectum next to it were diffusely red. In the small intestine, a segment of ileum had telescoped into another ileal segment near the ileocecal valve, forming an intussusception. The involved gut was swollen and, on cut surface, there was submucosal edema. Throughout the small intestine, there were small nodular areas which were raised and reddened. On histologic section, they consisted of a chronic inflammatory reaction resembling nodules associated with strongyle infection. The anterior mesenteric artery was thickened and firmer than normal; this is probably the mass that was palpated. The intussusception clouds the picture slightly but, because of the absence of passive congestion, it is believed that the intussusception developed when the animal was anesthetized or euthanatized and was not present prior to this. When we opened the anterior mesenteric artery, there was a mass of reddish brown friable material completely surrounding the wall of the artery, and only a very narrow lumen remained. On section, this mass had completely adhered to the vessel wall; in most areas, you couldn't differentiate the intima; and, in some instances, the thrombus dipped right down into the medial layer. In many areas,

*Dr. Fegley is associate in veterinary pathology.

there were chronic inflammatory cells: macrophages, plasma cells, and lymphocytes. In these areas of the thrombus, you could see the cross-sections of *Strongylus vulgaris*. Grossly, the rest of the necropsy was negative.

Dr. Brodey.—You felt that the portion of small intestine supplied by this anterior mesenteric artery showed no evidence of gross or microscopic changes?

Dr. Fegley.—I wouldn't say definitely about the microscopic sections because I haven't examined them that closely. As a matter of fact, I didn't take as many sections as I should have as most of the gut appeared grossly normal.

Dr. Brodey.—How does this tie in with malabsorption?

Dr. Marshak.—Since this was of long standing, it certainly could have been a malabsorption syndrome from a chronic ischemic condition of that part of the gut.

Dr. Mackay-Smith.—The clinician who performed the rectal examination on this horse wouldn't put his hand through a normal rectal wall. His comment at the time was that the rectum just seemed to fall apart.

Dr. Marshak.—A hypoproteinemia of that degree indicates tremendous tissue changes.

Dr. Mackay-Smith.—Microscopically, there would be less stroma if a protein depletion had been going on for a long time. I think that such protein structures as collagen would be in a weakened state, but I don't know whether you could appreciate this microscopically.

Dr. Sauer.—As far as the natural protein depletion is concerned, it usually affects the parenchymal cells themselves, and actually you get a relative increase in stroma. Another thing, speaking of this form of disease, we have to think, as Dr. Marshak says, from the functional rather than structural standpoint. One of the best examples of this is when a thrombus lodges in one of the iliac arteries. At rest, the tissue is maintained; it is only on exercise that clinical signs appear. I don't think it's at all impossible that a similar thing could happen in the bowel. If the bowel is deprived in this manner, you might not see any change in the muscle or the nerves microscopically, but many of the functions which the bowel has to perform could be upset.

Dr. Raker.—Dr. Fegley, we thought at necropsy that we saw a strongyle larva in one of the lymph glands. Have you had a chance to determine whether this is true?

Dr. Fegley.—There's no evidence of it in the sections that I've made.

*Dr. S. Dey.**—How about the liver? Was that changed at all?

Dr. Fegley.—Not grossly.

Dr. Marshak.—You didn't make sections of that?

Dr. Fegley.—No. I have spleen, lymph nodes, small intestine, colon, and artery.

Comment

*Dr. G. Graham.***—Speaking on behalf of the worm, I feel it is being forced to carry a bigger share of the responsibility here than it really deserves. I want to call your attention to one particular thing: namely, that there were no ova on fecal analysis. I'm curious to know why that should be and also to know how it can be, because I do not believe that an untreated horse will fail to have eggs of some sort in the feces. Perhaps there was a failure at the laboratory level or an indication of extensive, and more than adequate, treatment.

If phenothiazine had been used extensively, and that's the only anthelmintic which I understand is used presently for strongyles, it is conceivable that this colt, among other things, was overmedicated as far as an anthelmintic was concerned.

The type of lesion on the wall of the anterior mesenteric artery and the small hemorrhagic plaques on the wall of the small intestine are classical and are seen regularly. Those plaques are the points where larvae have drifted down in the circulatory system, lodged there, and caused erosion—a standard pattern. The lesion of the anterior mesenteric artery is one which essentially depicts a struggle between the host and the parasite. Here is a parasite that has to live in this part of the host for some length of time. Probably it has come in as a late third- or early fourth-stage worm. It has to attach itself to the wall of

* Dr. Dey is instructor in veterinary surgery.

**Dr. Graham is associate professor of parasitology.

the artery and hang on to pass through its molts. Then it lets go and undoubtedly is carried down and breaks through into the lumen of the gut and goes on to its normal habitat in the cecum. This type of lesion bespeaks a struggle of the host to overcome the parasite. I've seen similar histologic sections where some of the worms were being walled off, phagocytosed, and killed. The cuticle of the worm usually is not degraded by the enzymes of the horse, so it constitutes a foreign body, part of the debris that the horse keeps working on over and over again, and there is increased fibrosis. Ultimately there may be a rupture of the vessel.

From the standpoint of the essential biology involved here, I raise a question as to whether this is a full answer in this case. I do not believe that you have sufficient occlusion of the anterior mesenteric artery in this case to account for the full picture as we see it. Why don't you see this terminal picture routinely in all infected animals? Probably all animals are infected with this worm to a greater or lesser extent, conceivably even more heavily infected than this individual. Clinically, you would judge most of them to be essentially normal animals.

I don't know exactly what to say concerning the relationship of the worms to this particular case. That it has some relation I think we can accept; there's obvious damage there, but I do not think that the function of that artery was completely lost. This bespeaks a condition of long standing. The colt probably acquired some of the worms in Italy, others in England, and still others here. A great many horses live with these ubiquitous worms for a considerable time. This is a growing problem, and I am surprised that any race horses run on tracks anywhere, this worm problem being what it is.

The inability of this colt to achieve effective repair and destroy the worms may be related to the globulin deficiency, but I question whether the thrombus of itself is the primary disease; it may be a secondary rather than a primary factor.

Dr. Marshak.—Is it known that these worms selectively hang onto the mesenteric and iliac arteries or do they hang on all over?

Dr. Graham.—As far as we know, it's just in a fairly short area. I've seen early cases where there was almost no detectable damage; a little soft thrombus and tiny larvae that had not yet molted and were firmly attached to the wall. Five or six years ago, we had a specimen in which quite large worms had attached by the mouth-parts and actually had molted inside the vessel. They were adult male worms, which suggested, of course, that they had begun the fourth stage and molted at that point, and the only way they could get into the gut was to release their hold, be carried down into the circulation until they lodged, and then necrose the tissue there until they had eroded their way through into the lumen.

Dr. Marshak.—I wonder if you could tell us why they pick that segment of the gut?

Dr. Graham.—I've asked myself that many times without a satisfactory answer. The only comparable, or similar, case that I can mention is that of the giant kidney worm which is common in the mink and also infects dogs in certain localities. In areas where it is common in dogs (up to 35%), you invariably find the parasite in the right kidney; if it is not in the right kidney, it's in the abdomen and the right kidney is destroyed. Now if you can explain why this worm goes into the right kidney, I'll try to explain why the strongyle worms localize in the anterior mesenteric artery.

Dr. Mackay-Smith.—With respect to your question, Dr. Graham, about whether the colt had been treated, I think it's likely that he had been. It is the custom in this part of the country to "worm" all new animals. This animal came into our clinic about two weeks after he had landed in this country, so I feel sure that he was given a dose of phenothiazine a couple of weeks before.

Dr. Graham.—I know it's routine practice. One of the quarrels I've always had concerning low level phenothiazine treatment is that one of the mother substances of phenothiazine, diphenylamine, is a dehydrogenase inhibitor, and I suspect that this interferes rather seriously with the whole microbial flora in the horse's lower bowel. If you're giving an animal constant, steady doses of this medicament, you may be seriously interfering with his digestion and with his absorption of foodstuffs, as well. You know we can't be quite sure just what we are doing—certainly we're not

getting rid of the worms, although low-level phenothiazine does interfere rather seriously with the worms as far as their ability to produce viable eggs is concerned. It still doesn't keep the worm from getting along after a fashion.

Dr. Marshak.—Do they actually hang onto the wall, or do they burrow into the wall?

Dr. Graham.—They attach themselves.

Dr. Marshak.—With a sucker of some sort? How much of a lesion do they produce on the arterial wall?

Dr. Graham.—It's still a matter of argument. The enzymes in the secretions of the worms' esophageal glands probably form a clotlike mass of fibrin there so that they're hanging free, just floating in the stream. The flow through the vessel is strong enough to carry away rather rapidly anything that isn't anchored. The fact that they are able to locate themselves there means that they have to take hold rather quickly if they're going to stay at all. It may be that it's necessary for them to hang on there to transform. We don't know whether those that are able to attach themselves are the ones that establish themselves in the gut, or whether it's the ones that are swept right on down that enter the gut. There are a lot of blind spots insofar as our knowledge of the life cycle of these worms is concerned. I think all horses are exposed to a greater or lesser extent. It's something they ordinarily live with, but here is one that didn't live with it well.

Dr. Mackay-Smith.—Would I be representing a summary view if I say that this animal probably died of a thrombus of the mesenteric artery, but that the predisposing factors and incidents in its biologic existence may have been much more complex and much further removed than that? We probably won't know a great deal more about them until the next case we see, and maybe not then.

Pathologic Diagnosis

1) Thrombosis of the anterior mesenteric artery, secondary to strongylosis.

2) Rupture of the rectum, with diffuse peritonitis.

3) Terminal intussusception of the ileum.

Addendum

*Dr. W. D. Malherbe.**—There is considerable doubt in my mind as to the quantitative accuracy of the albumin globulin ratios given here. Total protein would be correct, but it was found after this conference that the technician concerned had erred in manipulating the reagents in this procedure. The 22.8% sodium sulfate globulin precipitate previously used in our laboratory had been discontinued in favor of 28.0% sodium sulfite, and the efficiency of ether separation had been enhanced by the addition of a detergent and caprylic alcohol, giving figures in closer accord with those obtained by electrophoresis.

*Dr. Malherbe is visiting professor of clinical laboratory medicine.

Parakeratosis in Swine Has Complex Origin

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In one trial, some pigs fed rations low in zinc and given city water developed parakeratosis while those given well water did not.—*Feedstuffs, 33, (Sept. 30, 1961): 26.*

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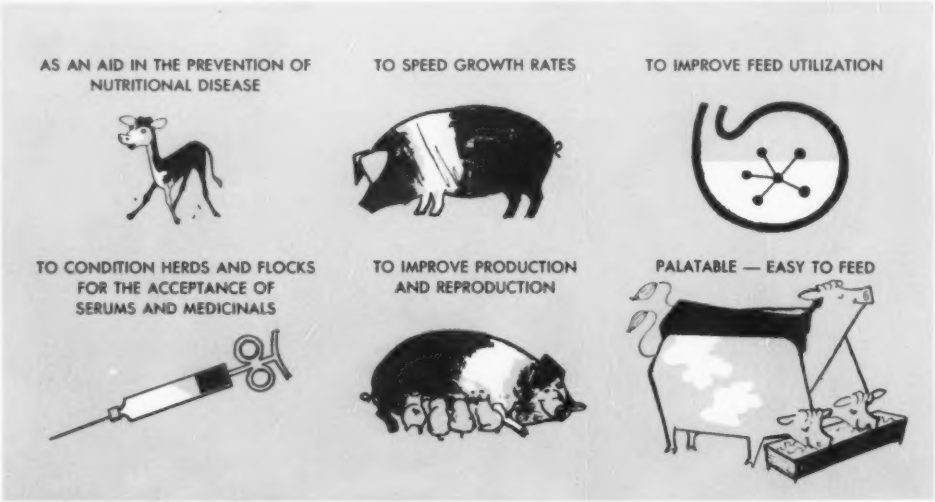
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The educational campaign to farmers on behalf of the profession is now being carried on through farm magazines, newspapers, television, and radio.

Member companies who are underwriting the campaign by the American Foundation for Animal Health include: Allied Laboratories, Inc., Blue Cross Serum Co., Corn Belt Laboratories, Inc., Corn States Laboratories, Inc., Fort Dodge Laboratories, Grain Belt Supply Co., Haver-Lockhart Laboratories, Liberty Laboratories, Norden Laboratories, Pitman-Moore Co., Sioux Falls Laboratories, Inc., Southwestern Laboratories, Inc., the Gregory Laboratory, Inc., and the National Laboratories, Corp.

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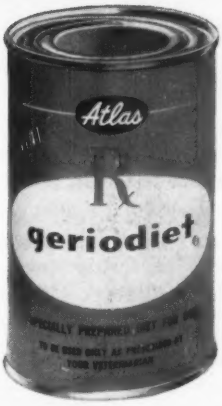

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COUNCIL REPORT

AVMA Council on Public Health and Regulatory Veterinary Medicine

Bovine Tuberculosis

The instruments, scientific principles, methods, and procedures for diagnosis and elimination of bovine tuberculosis have remained essentially unchanged since introduction of the eradication program over 40 years ago. Last year, 88 tuberculous cattle carcasses were condemned by federal meat inspection officials. During the previous year, 91 carcasses were condemned; 114 the year before that; and in 1917, a total of 49,214.

TB Infection and the Tuberculin Test

These results have been achieved primarily through use of the intradermal tuberculin test. This test, properly applied, is still our best diagnostic tool, but the relative cost in man hours, funds, and cattle increases sharply as we approach zero incidence of infection. Factors affecting the tuberculin test, of no practical significance in early phases of the program, are assuming such proportions today that further progress without benefit of more discriminatory diagnostic products and more meticulous techniques is rapidly approaching the limit of economic tolerance.

Last year, 14,000 reactors to the tuberculin test were removed from herds in this country. Approximately 70% of that number did not have gross lesions of tuberculosis on necropsy. Of the reactors with no gross lesions, 14% were in known infected herds and therefore may be discounted, as they have been in the past; and, in some instances, careful laboratory examinations have disclosed bovine tuberculosis in no-gross-lesion reactors from no-gross-lesion herds. On the other hand, all available evidence today indicates that the incidence of *Mycobacterium bovis* infection in the United States is considerably below that of *M. tuberculosis* or *M. avium*. Tuberculosis, in spite of modern therapeutic measures, is the greatest single microbial killer of man. Almost half the human population in the United States in the 60 to 64 year age group reacts to the Mantoux test and represents potentially active cases. Approximately 3% of swine slaughtered in federally inspected establishments have lesions of tuberculosis, almost exclusively of the avian type. Tuberculosis is still prevalent in mature poultry in some areas. According to federal poultry inspection reports, 0.6% of all mature chickens slaughtered in the west northcentral states are condemned for tuberculosis, and the same disease accounts for 28% of total condemnations for disease.

These other types of mycobacterial infection, to which cattle are exposed, present a progressively greater threat to our livestock industry, not in the form of clinical diseases but by hypersensitization to tuberculin, our diagnostic agent. Field studies now in progress tend to confirm the theory that other acid-fast bacteria are also a factor in tuberculin hypersensitization. This problem is recognized by Dr. William H. Feldman, who states: "One of the most urgent and potentially fruitful problems for research in tuberculosis of man and animals is that embracing the reciprocal relationship of the tuberculin reaction to hypersensitivity induced by mycobacteria other than true tubercle bacilli."

Review of Accreditation Requirements

Counties continue to be reaccredited under the provisions of the Uniform Methods and Rules for Bovine Tuberculosis Eradication, but reaccreditation requirements

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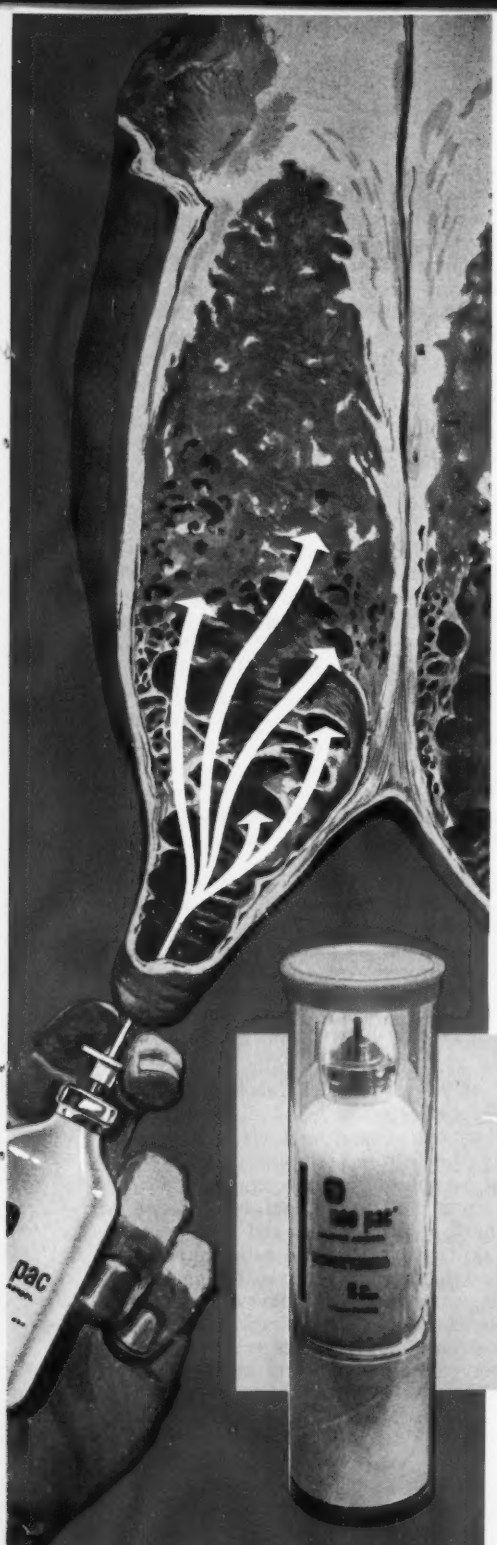

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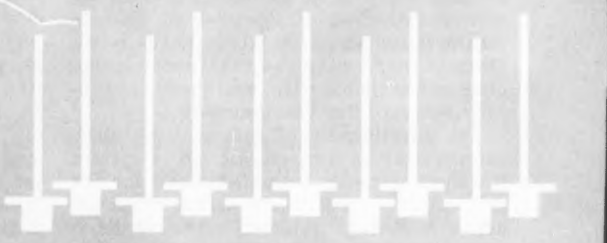

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are under constant review and evaluation to assure that these requirements serve the best interests of disease eradication.

We continue to test less than 10% of the cattle population annually, although every effort is made to concentrate such testing in those areas and in those herds with the greatest potential for infection.

Evaluation of New Procedures

Field tests are now in progress for the purpose of developing, if possible, an effective tuberculin screening test to identify more infected herds and at the same time eliminate a significant percentage of tuberculin reactions caused by mycobacteria other than *M. bovis*. Effective utilization of intensive eradication procedures in known infected herds will reduce incidence of tuberculosis in cattle considerably. This phase of the program is dependent to a large degree upon the thoroughness and effectiveness with which infected premises are cleaned and disinfected. This is a weak link in the eradication program, and one deserving of an all-out effort to correct. Preliminary cleaning, more important by far than disinfecting alone, is delegated to the owner, who frequently has no conception of the significance of this disagreeable, tedious job.

The least that we, as accredited or regulatory veterinarians, can do is to inform the owner fully concerning the absolute necessity of a thorough cleaning job, in his own interest and as his contribution to the over-all reduction of this disease. Serious consideration should be given to the possibility of performing the work under immediate supervision with equipment specifically designed for that purpose.

The effectiveness of comparative tuberculin testing as a diagnostic aid in certain instances is being evaluated. Need for the eradication of *M. tuberculosis* and *M. avium* is obvious. Intensifying tuberculosis eradication efforts in other animals and poultry concurrently with the bovine tuberculosis eradication program is desirable and necessary. The bovine subject cannot be considered a persistent reservoir of infection for these types of mycobacteria, however. Consequently, we must consider *M. bovis* primarily when applying diagnostic tuberculin tests to cattle. Attempts to develop an accurate and practical serologic test for tuberculosis in livestock have met with little success thus far, but work is still being carried on in this field.

Public Health

Human tuberculosis of the bovine type has almost disappeared in the United States. During the past decade, less than 12 confirmed human infections have occurred. The last proved case in a child was in 1956, and in an adult, 1958. This in itself is a tribute to the control of bovine tuberculosis.

Perfection and Expansion of Known Effective Procedures

Epizootiologic studies are conducted on problem herds and case-finding procedures are being perfected and expanded. Laboratory assistance is being intensified, and necropsy procedures at slaughtering establishments under inspection systems other than federal are being brought into the program. Tracing procedures already led us to 7% of all tuberculin reactors and 19% of all gross lesion reactors.

While efforts are being directed toward development of sharper tools and more effective procedures, the success of the program depends to a large degree upon getting complete and factual information to the herd or flock owner and upon the care and skill with which the tuberculin test is applied. There is a renewed national interest in, and concern for, this disease. There is need for sober consideration and sincere dedication by all of us, as veterinarians, to do our part in conquering tuberculosis.

Instructions to Authors

JOURNAL *of the AVMA*

1) *Exclusive Publication*: Articles submitted for publication in the JOURNAL of the AVMA are accepted with the understanding that they are not being offered for publication elsewhere.

2) *Manuscripts*, including footnotes, references, tables, and legends, must be typewritten, double-spaced, on 8½- by 11-inch bond paper, and the original and one carbon copy should be submitted. One-inch margins should be allowed on the sides with 2-inch margins at the top and bottom. Articles should be concise.

Purpose of the article should be stated in the introduction. Summary should be included.

3) *References* to published works should be brief and limited strictly to what is relevant to satisfactory exposition of the author's own work. Reference names are not permitted in the text of the article. Persons cited are to be referred to by superscript numbers only, relating to the reference list at the end of the article.

References should be typed double-space, in alphabetical order by author, as follows: author(s), title, periodical name, volume, year, and page(s).

[5]Mansson, J., and Obel, N.: The Technique of Adrenalectomy in the Ruminant. Cornell Vet., 48, (1958): 197-201.

When books are cited, name of publisher, location, edition, year of publication, and pages concerned should be given.

[1]Arey, L. B.: Developmental Anatomy. 5th ed. W. B. Saunders Co., Philadelphia, Pa. (1946): 236-240.

4) *Tradenames* of products are avoided in the text of the article. Use only generic or chemical names. The tradename may be included in a footnote along with name and location of producer (composition of product may be included if necessary): e.g., ". . . piperazine citrate* . . ." Footnote: *Piperol, Carson Chemicals, New Castle, Ind.

5) *Arabic numerals* should be used wherever digits are needed throughout the article except at the beginning of a sentence.

6) *Abbreviations* for weights and measures as given in standard dictionaries are usually acceptable, but some are determined by AVMA editorial preferences: e.g., Gm., gr., kg., cc., mg., cmm., Gm./kg., μg., mg./100 ml. Other abbreviations should be explained the first time they are mentioned either in parentheses or footnotes: e.g., ". . . pregnant mare's serum gonadotrophin (PMSG). . ."

7) *Photographs* should be furnished in glossy prints. Identifying arrows, letters, etc. within photographs should be clearly defined. All illustrations should bear the author's name, illustration number, and "top" side indication.

8) *Drawings*, graphs, and charts should be clear and large enough to allow for possible reduction in size. A glossy print should be submitted if possible.

9) *Tables* should be simple and typed double-space. Complex tables are not acceptable. Complex material should be summarized rather than tabulated.

10) *Legends* should be typed double-space on a separate sheet.

Sufficient information should be included to allow the illustration to be understood without reference to the text.

Vitamin A Deficiency in Cattle

During the past 3 years, there has been an increasing number of diagnoses of vitamin A deficiency in fattening cattle on rations formerly considered to supply amounts of carotene in ample excess of the animal's requirements. Many field diagnoses of such deficiency have been based on the appearance of lameness in a relatively small percentage of individuals and on subsequent recovery from this lameness following injections of vitamin A by practicing veterinarians. Recovery from lameness following injections of vitamin A does not necessarily warrant a diagnosis of vitamin A deficiency. Supporting evidence such as blood and liver vitamin A assays are also necessary to confirm a diagnosis.

The increase in incidence of vitamin A deficiency may be due to excessive nitrate concentrations in green forages. Nitrates interfere with carotene and vitamin A utilization.—*Nutri. News Bull. (Oct., 1960): 1-4.*

Dr. B. Scott Fritz Retires

Dr. B. Scott Fritz (UP '17), former managing director and assistant vice-president of Wyeth Laboratories at Marietta, Pa., has retired after 26 years with the firm.

After serving in the U.S. Army from 1917 to 1920, where he became a major in the Veterinary Corps, Dr. Fritz conducted veterinary research at the University of Pennsylvania. From 1927 to 1935 he worked for the Pennsylvania Bureau of Animal Industry. He then joined the Wyeth staff.

Coming Meetings

Notices of coming meetings must be received 30 days before date of publication.

American Animal Hospital Association. Regional Meetings. Dr. Frank R. Booth, 3920 E. Jackson Blvd., Elkhart, Ind., executive secretary.

Region 2. Hotel Patten, Chattanooga, Tenn., Feb. 11-12, 1962.

Region 3. Secor Hotel, Toledo, Ohio, March 26-27, 1962.

Region 4. Mayo Hotel, Tulsa, Okla., Jan. 20, 1962. Texas Hotel, Fort Worth, Texas, Feb. 4, 1962. Colorado State University, Fort Collins, Colo., Feb. 18, 1962.

Region 5. Sheraton Meeting House, Honolulu, Hawaii, May 16-17, 1962.

November, 1961

Arizona Veterinary Medical Association. Annual meeting. Pioneer Hotel, Tucson, Ariz., Nov 26-28, 1961. Dr. C. E. McGlamery, 3627 E. Grant Rd., Tucson, Ariz., publicity chairman.

December, 1961

American Association of Equine Practitioners. 8th annual meeting. Hotel Texas, Fort Worth, Texas, Dec. 3-5, 1961. AAEP, 531 Guaranty Bank Bldg., Denver 2, Colo.

Kentucky Veterinary Medical Association. Annual meeting. Kentucky Hotel, Louisville, Ky., Dec. 4-5, 1961. Dr. L. S. Shirrell, 545 E. Main St., Frankfort, Ky., secretary.

Nebraska Veterinary Medical Association, Inc. Annual convention. Hotel Cornhusker, Lincoln, Neb., Dec. 5-7, 1961. Mr. Bob Garey, Office Rooms 418-419, Hotel Clarke, Hastings, Neb., executive secretary.

January, 1962

Cornell University. Conference for Veterinarians. Cornell University, New York State Veterinary College, Ithaca, N.Y., Jan. 10-12, 1962. Dr. Peter Olafson, Department of Pathology, Cornell University, Ithaca, N.Y., program chairman.

University of Pennsylvania. Sixty-second annual conference of veterinarians. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa., Jan. 16-17, 1962. Dr. Robert Schwartzman, program chairman.

Michigan State University. Thirty-ninth annual Postgraduate Conference for Veterinarians. College of Veterinary Medicine, Michigan State University, East Lansing, Mich., Jan. 17-19, 1962. Dr. W. W. Armistead, dean.

Tennessee Veterinary Medical Association. Annual meeting. Dinkler-Andrew Jackson Hotel, Nashville, Tenn., Jan. 21-23, 1962. Dr. H. W. Hayes, 5009 Clinton Pike, Knoxville, Tenn., secretary.

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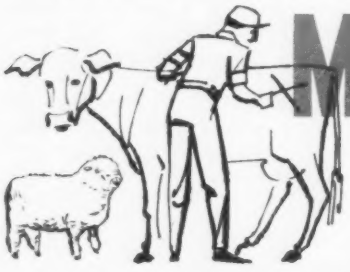

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Oklahoma Veterinary Medical Association. Annual meeting. Mayo Hotel, Tulsa, Okla., Jan. 21-23, 1962. Dr. W. D. Speer, 538 S. Madison, Tulsa, Okla., secretary.

Minnesota Veterinary Medical Association. Annual meeting. Hotel St. Paul, St. Paul, Minn., Jan. 22-24, 1962. Dr. B. S. Pomeroy, 1443 Raymond Ave., St. Paul 8, Minn., secretary.

Intermountain Veterinary Medical Association. Annual meeting. Newhouse Hotel, Salt Lake City, Utah, Jan. 24-27, 1962. Dr. Bert Reinow, Box 277, Pinedale, Wyo., president.

North Carolina Veterinary Conference. North Carolina State College, Raleigh, N. Car., Jan. 24-25, 1962. Dr. E. G. Batte, Head, Veterinary Section, School of Agriculture, North Carolina State College, Raleigh, N. Car., program chairman.

Mississippi State Veterinary Medical Association, Inc. Hotel Heidelberg, Jaskson, Miss., Jan. 27, 1962. Dr. W. H. Lindley, Box 444, Vicksburg, Miss., secretary.

Kansas Veterinary Medical Association. Annual meeting. Broadview Hotel, Wichita, Kan., Jan. 29-31, 1962. Dr. M. W. Osburn, 1525 Humboldt, Manhattan, Kan., executive secretary.

February, 1962

Nevada State Veterinary Association. Annual winter meeting. Holiday Hotel, Reno, Nev., Feb. 12-13, 1962. Dr. Brian L. Hutcherson, 300 Kietzke Lane, Reno, Nev., secretary.

Arkansas Veterinary Medical Association. Forty-fifth annual meeting. Hotel Marion, Little Rock, Ark., Feb. 18-20, 1962. Dr. T. D. Hendrickson, 7824 Cantrell Rd., Little Rock, Ark., secretary.

August, 1962

American Veterinary Medical Association. Ninety-ninth Annual Meeting. Fontainebleau Hotel, Miami Beach, Fla., Aug. 12-16, 1962. Dr. H. E. Kingman, Jr., 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Foreign Meetings

Twelfth World's Poultry Congress. Show Grounds of the New South Wales Royal Agricultural Society, Sydney, Australia, Aug. 13-18, 1962. Dr. Cliff D. Carpenter, chairman, U.S. Participation Committee, 1207 Emerald Bay, Laguna Beach, Calif.; Dr. A. William Jasper, secretary, c/o AFBF, 2300 Merchandise Mart, Chicago 54, Ill.

AVMA Research Fellowships Available

The Council on Research of the AVMA announces the availability of a number of fellowships for postgraduate training for the academic year, 1962-1963.

The recipient of a fellowship must be a veterinarian and a citizen of the United States or Canada. Veterinary students who expect to graduate at the end of the current school year and who wish to follow a career in research may apply for a fellowship.

It is advisable that completed application forms be filed by Jan. 1, 1962, to allow time for correcting omissions and for some reasonable delay in arrival of letters of information from third parties. The latest date for filing the completed application is Feb. 1, 1962. Approximately one month is required for processing completed applications after receipt by the Council. Qualified persons should secure and submit applications as early as possible to insure their file being complete for presentation to the Committee on Fellowships.

The Committee on Fellowships of the Council on Research will meet early in March to consider applications and the awards will be announced soon afterward. The stipend will be determined in each case by the needs of the individual, the location of the school in which he proposes to work, and other factors. In general, the stipends range from $100 per month upward. Any qualified person interested in graduate training may obtain application blanks and other information from the deans of the various colleges of veterinary medicine or by writing to the Council on Research, American Veterinary Medical Association, 600 S. Michigan Ave., Chicago 5, Ill.

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Are relapses a problem?

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History of the AVMA

1922

In his presidential address at the 1922 meeting in St. Louis, A. T. Kinsley said, "Until about 1912, practitioners were primarily engaged in the medical or surgical relief of disease . . . . Equine practice constituted the major portion of . . . . routine business . . . . Diseases of cattle . . . . swine and poultry were rarely given more than a passing thought . . . . The veterinarians services at this time are principally confined to . . . . diseases of meat-producing animals in herd units . . . . The successful veterinarian of today is an immunologist; . . . . of 20 years ago . . . . a surgeon . . . . This transition was too sudden and complete for the best interests of all concerned.

Recognizing the retrenchment of the profession as a result of the depression, Dr. Kinsley observed that its future would be dependent on relations with agricultural interests. "The veterinary profession will survive, because . . . . [it] alone insures the livestock industry against the ravages of disease and is indispensable [for] . . . . the health of nations." Earlier, N. S. Mayo had pointed out that the depression had affected everyone, financially and psychologically, and that many veterinarians had given up their profession for other work.

Speaking on "The Trend of Veterinary Practice," W. H. Welch said, ". . . the graduate . . . . is no longer just a 'horse doctor,' but is the confidential adviser of the livestock owner concerning all the domesticated animals . . . . As a sanitarian he is no less valuable than the physician in guarding the health of his client's family. He is the leader in bacteriology, pathology and other fields of original research, while as inspector of foods his services are indispensable."

Urging that veterinarians adopt a positive program to deal with the problem of lay vaccination of hogs, Clarence Ousley, former Assistant Secretary of Agriculture, stated, "A veterinarian is something of a missionary, and he is warranted in doing something more than sitting on his dignity and awaiting calls from an uninformed populace."

For the first time, a session was devoted entirely to small animal practice, with reports by O. V. Brumley, H. J. Milks, M. C. Hall, R. P. Marsteller, W. E. Muldoon, and G. P. Frost. The cesarean operation was demonstrated by W. G. Brook, with intratracheal anesthesia by J. G. Hardenbergh, who also spoke on the value of animal experimentation to veterinary medicine. Other reports included those by W. Giltner, I. F. Huddleson, R. A. Kelser, W. E. Cotton, R. R. Birch, A. F. Schalk, W. L. Boyd, R. J. Foster, V. A. Moore, and Drs. Dorset, McBryde, and Niles.

H. P. Hoskins was appointed to the full-time office of combination secretary and editor.

ALBERT THOMAS KINSLEY was born at Independence, Iowa, Feb. 26, 1877, received his B.S. (1899) and M.S. (1901) degrees at Kansas State, studied pathology at the University of Chicago, and graduated from the Kansas City Veterinary College in 1904. A successful practitioner, he purchased an interest in the latter school in 1912, became its president, and taught pathology, bacteriology, and parasitology. He contributed frequently to the

Dr. A. T. Kinsley

veterinary literature, and pioneered a section on swine practice in *Veterinary Medicine* when this specialty was little known outside the midwest. He was widely known as a skilled diagnostician, a manufacturer of veterinary biological products, and an exponent of better veterinary education. He was the author of two books, *Swine Practice*, and *Textbook of Veterinary Pathology*.

Dr. Kinsley was active in the Missouri Valley Veterinary Association, and he served the AVMA in numerous capacities, including the presidency (1921-22). He died on Dec. 8, 1941.

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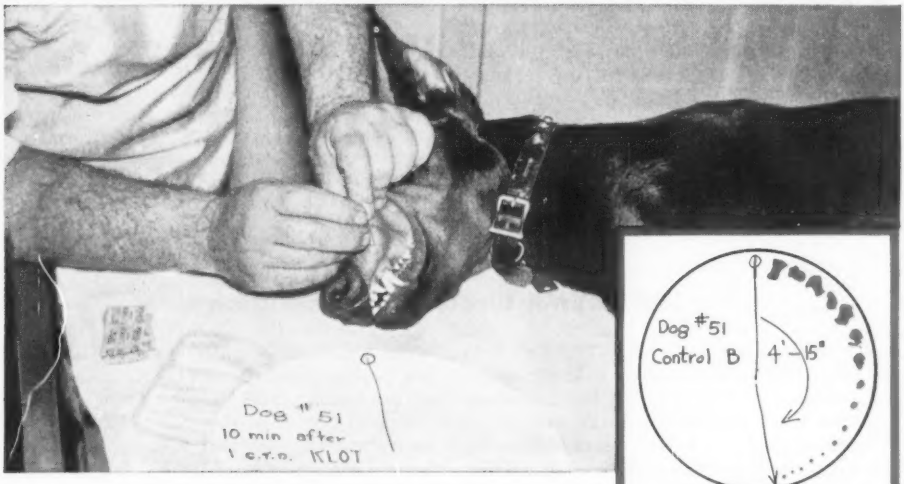

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Public Relations

1962 AVMA Directory in Production

The coming year will be a Directory year for the AVMA. Biannually, the central staff concentrates on the massive job of collecting the vital statistics on the veterinary profession and compiling a comprehensive listing of veterinarians. With each edition, this task has become a more challenging one. The 1960 edition listed approximately 16,500 members of the profession; the 1962 issue will list more than 23,000 veterinarians, including those who are not members of the AVMA.

Automated techniques will assist in handling the task more efficiently. New data processing machinery installed in November will write the listings for the Directory as its first assignment. Listings prepared by the equipment from punched cards will be photographed, and printing plates made for the production of the edition. Everything about the new volume will be bigger; the type size used in the listings will be larger; the over-all dimensions of the book itself will be increased to 8½ by 11 inches; and, since more individuals will be listed, the total page length will increase considerably. If present plans prevail, the staff work now in progress should meet its fruition in early 1962 in a more comprehensive and accurate edition than ever before produced.

One major job is now completed. In a series of mailings, the AVMA attempted to verify every address and all of the statistical data which would go into the Directory. In the past, each individual was listed in two sections of the volume—the geographic listing (by states) which gave the individual's name and full address along with coded information which identified his professional specialty, school, and year of graduation, and the alphabetical portion which listed only the name and state of residence of each veterinarian in alphabetical sequence.

The mailings, in which each veterinarian was asked to verify his name and address, specialty, and school and year of graduation created an unusual amount of membership reaction in regard to the data which would be listed. The AVMA uses a coding system for classifying the professional specialties of veterinarians designed by the National Science Foundation for all individuals engaged in work in the health field. These codes are helpful in many ways. Not only do they provide easy "handles" for dealing with many types of veterinary activity, but their uniformity with other health professions provides a valuable working tool for all who are interested in total health manpower resources. The codes did not meet with universal approval among members of the profession, however, since they did not adequately identify several important areas of veterinary activity. Teachers, research workers, and industrial veterinarians all end up, in the present system, as code 2599, or "other veterinary medicine."

Cognizant of the inadequacies of the coding system, the AVMA will add other codes to the specialty code information in the new edition. For some years, codes have been maintained on each individual's type of employer and employment function. Though they have not been printed before, nor have they been verified in the current

mailings, these codes will appear in the 1962 edition. The new codes, added to the information given in the specialty code information, will enhance the profile of each veterinarian and make the book more informative and useful.

In addition, an identifying symbol will be printed for each diplomate of a specialty board recognized by the AVMA. Presently, there are three such organizations: The American Board of Veterinary Public Health, The American College of Laboratory Animal Medicine, and The American College of Veterinary Pathologists.

In the new Directory, the geographic section will list only the name and full address of the individual. In the alphabetical section, each veterinarian's name will appear along with the state in which he resides, his professional specialty code, school, year of graduation, employment type, employment function, and specialty organization if any.

Some reaction is expected to the addition of unverified information in the new edition; however, it is hoped that, by adding this information, the awareness of the membership of the types of information which the AVMA maintains can be increased, and that each member will take it upon himself to supply the central office with correct data from time to time.

The Directory is scheduled to go into full production this month, and the announcement of its completion is expected in late January or early February of 1962.

R. D. MORRISON

Director of Membership Services

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15th of month issue — 22nd of month preceding date of issue.

Names of classified advertisers using key letters can not be supplied. Address your reply to the box number, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be sent to the advertiser.

Wanted—Veterinarians

Wanted—experienced veterinarian with California license for an excellent opportunity with California institution. Address Box L 63, JOURNAL of the AVMA.

Wanted—veterinarian as associate in AAHA member hospital. Salary depending upon experience. Please state qualifications and availability in first letter. Michigan license required. Address Box L 62, JOURNAL of the AVMA.

Wanted—recent graduate interested in thriving mixed practice, 60% small and 40% large. Modern small animal hospital near large city in southern Ontario. Opportunity for advancement to full partnership. Address Box L 61, JOURNAL of the AVMA.

Wanted—veterinarian for permanent position in AAHA hospital in Washington, D. C. suburbs for small animal practice. Good salary, working conditions. Send full details. Address Box L 60, JOURNAL of the AVMA.

Wanted—associate veterinarian for small animal hospital in New York City suburb. Must be experienced and have license. Salary—$10,000 up. Excellent opportunity for ambitious person. Address Box L 54, JOURNAL of the AVMA.

Oregon Department of Agriculture

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Wanted—veterinarian for small animal practice in Queens, N. Y. Top salary and excellent working conditions. Permanent position. Address Box L 52, JOURNAL of the AVMA.

Wanted—associate veterinarian for small animal hospital in Westchester County, N. Y. Top salary and excellent working conditions. Address Box J 16, JOURNAL of the AVMA.

Wanted—associate veterinarian, preferably with experience in laboratory work, for AAHA hospital in Missouri. Complete details in first letter. Address Box L 17, JOURNAL of the AVMA.

Wanted—veterinarians interested in teaching and research in physiology or pharmacology at a veterinary college. Opportunities for graduate work. Address Box L 16, JOURNAL of the AVMA.

Wanted—Positions

Veterinarian, age 26, service completed, desires association with progressive small animal practice in New Mexico, Colorado or Texas. Address Box L 66, JOURNAL of the AVMA.

Graduate (MSU '61) with Florida license desires employment in small animal practice in Southern Florida area but will consider other areas in Florida. Address Box L 58, JOURNAL of the AVMA.

Graduate veterinarian, University of Bogota, Columbia, S. A., familiar with large and small animals, willing to work under your supervision in any capacity. Unable to secure license until naturalized. Age 47, health excellent, can read and write English. Will relocate. Present employer will furnish references. Address Vicente Escobar, 240 Godwin Ave., Midland Park, N. J.

Experienced veterinarian desires position in public health administration, teaching and/or research. Education: M.P.H., D.V.M. Military obligation completed. Address Box L 57, JOURNAL of the AVMA.

Energetic young veterinarian, MSU graduate, experienced in small animal practice and executive hospital management, military obligation completed, desires position in progressive practice in Midwest. Will consider lease arrangement. Address Box L 56, JOURNAL of the AVMA.

Young veterinarian with references from veterinarian employers desires position with future in a Texas small animal practice. Address Box L 55, JOURNAL of the AVMA.

Experienced, licensed veterinarian available for regular part-time work in Westchester, Bronx, or southern Connecticut. Address Box L 53, JOURNAL of the AVMA.

Recent graduate with 2 years' experience in large AAHA hospital desires position, lease, or purchase of small animal hospital in East or Southeast. Address Box L 12, JOURNAL of the AVMA.

Veterinarian's assistant and receptionist, thoroughly experienced, excellent references, wants live-in position. Widow, aged 45, salary wanted—$50 per week. Prefer southern location. Address Mrs. Cecil M. Cross, General Delivery, Jonesboro, Ark.

For Sale or Lease—Practices

For sale—one of Florida's best, well-established, well-equipped, lucrative small animal hospital and practice. Price—$85,000. $25,000 required to handle. Address Box L 67, JOURNAL of the AVMA.

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For sale or lease—northeastern Indiana hospital, fully-equipped for large and small animals including chute and x-ray. Good mixed practice netting more than $10,000. Available immediately. Large frontage on state highway. Address Kenneth Springer, Ossian, Ind., or Dr. K. M. Weinland, 327 W. Lutz Ave., West Lafayette, Ind.

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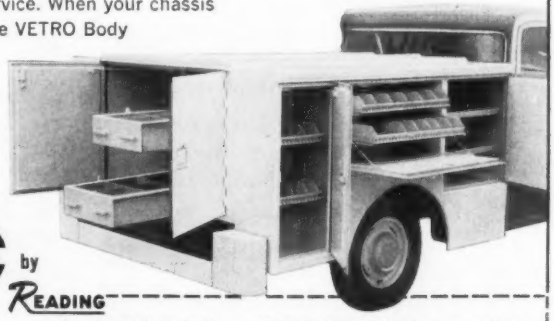

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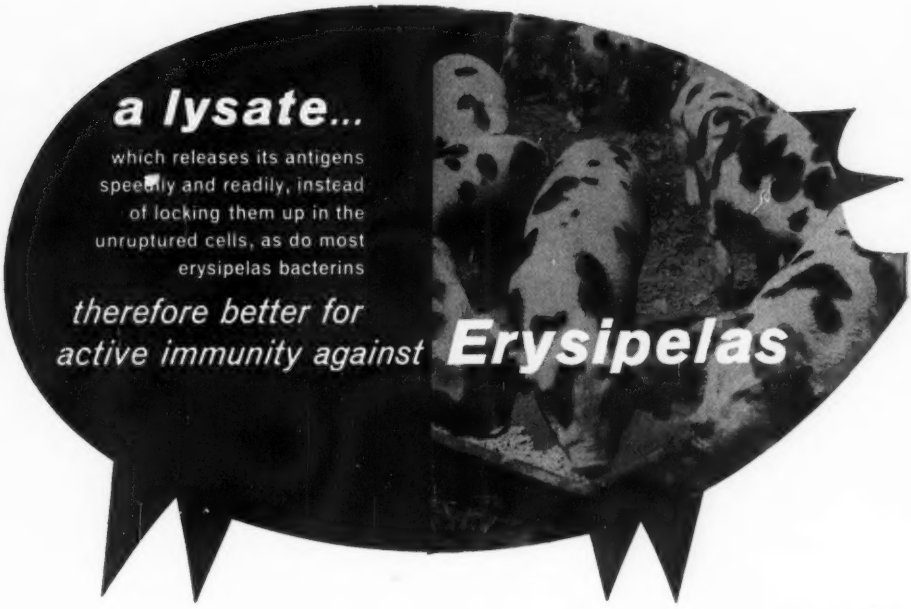
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1. Sinha, S. K., et al, Studies on Canine Distemper Immunization with a Tissue Culture Vaccine. Vet. Med., Vol. 55, No. 4, April 1960.
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